The Journal of Thoracic Surgery

VOL. 35 JANUARY, 1958 No. 1

RUDOLPH MATAS

The Editorial Board notes with profound sorrow the death on September 23, 1957, at the age of 97, of Dr. Rudolph Matas, a charter member and third president of The American Association for Thoracic Surgery and a member, from its inception in 1932, of the Advisory Editorial Board of the JOURNAL OF THORACIC SURGERY. Thus passes from the American and international scene a great figure in Surgery, respected, revered, and beloved by all fortunate enough to have known him during his long and distinguished career. It can be said of Dr. Matas that in the depth of his medical and surgical knowledge, in the facility with which he presented that knowledge to others, in several languages, if need be, and in the beautiful style of his spoken and written word, he was truly unique, unequaled, and unexcelled. His loss as a valued associate on the Editorial Board and as a sympathetic and loyal friend is mourned by all his fellow members on the Board.

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Original Communications

RECONSTRUCTION OF THE ESOPHAGUS WITH SEGMENTS OF THE COLON

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ESOPHAGEAL replacement utilizing the colon to create an antethoracic subcutaneous esophagus was first used by Kelling and Hacker in the early 1900's. Although, in a review of the literature from 1911 to 1934, Ochsner found only 20 cases of subcutaneous colon transplants,[15] in the past few years there has been a revival of this method in Europe. Our unsatisfactory results in using the stomach to restore alimentary continuity, following resection for carcinoma, led us to investigate the technique of replacing the esophagus with the colon.

It is the purpose of this paper to present our clinical and experimental observations on the efficacy of using segments of the colon within the thorax to restore esophageal continuity following resection in both benign and malignant disease. While this over-all approach to reconstructing the esophagus is not new, certain recent advances of medicine appear to make this a reasonable approach to this intriguing problem.

EXPERIMENTAL PROCEDURE

To evaluate the reconstruction of the esophagus by implanted colon, a series of 6 normal dogs were operated upon. Through a thoracoabdominal incision, the esophagus was resected from the inferior pulmonary vein to the cardioesophageal junction. The opening in the stomach was inverted. An appropriate segment of transverse colon was interpolated in isoperistaltic fashion between the esophagus above and a separate opening in the fundus of the stomach. A colocolostomy reconstituted the colon. Pyloromyotomy or pyloroplasty provided adequate drainage of the vagotomized stomach. These dogs were sacrificed at varying periods up to 9 months following surgery. In a comparative series, 5 dogs had a resection of the distal end of the esophagus and an esophagogastrostomy performed. Bilateral vagotomy and pyloromyotomy or pyloroplasty were done. The diaphragm was sutured to the stomach so that a considerable portion of the latter lay well within the thorax. These dogs were then sacrificed after varying periods of time up to 3 months following surgery.

EXPERIMENTAL RESULTS

The lower end of the esophagus, the transplanted colon, and the entire stomach were removed for post-mortem examination. In none of the animals

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Read at the Thirty-seventh Annual Meeting of the American Association for Thoracic Surgery at Chicago, Ill., May 4-7, 1957.

in this group was there evidence of esophagitis, colitis, or gastritis (Fig. 1). Post-mortem examination in the second group of animals showed complete absence of the mucosa in the lower end of the esophagus above the esophagogastric junction. There were 3 instances of diffuse ulceration of the fundus which lay within the thoracic cavity. The mucosa for the most part, extending up to 3 to 4 inches above the cardioesophageal junction, was markedly flattened with evidence of esophagitis and ulceration (Fig. 2).

Fig. 1.

Fig. 2.

Fig. 1.—Post-mortem examination of esophagus, interposed colon, and stomach in a dog sacrificed at 9 months. There is no evidence of esophagitis, colitis, or gastritis.

Fig. 2.—Post-mortem examination following an esophagogastrostomy. There was diffuse inflammation in the stomach and ulceration of the esophageal mucosa.

CLINICAL OPERATIVE TECHNIQUE

The esophagus was reconstructed in one stage with the exception of one child in whom a delayed reconstruction was performed at the age of one year. Two separate operating teams have been employed. Satisfactory preoperative nutrition and chemical balance were obtained by either preliminary feedings through a small polyethylene tube passed into the stomach at the time of the esophagoscopy or a tube gastrostomy. The colon was prepared for 2 to 4 days with neomycin (1 Gm., 4 times daily), Fleets phosphosoda (½ oz., twice

daily), and cleansing enemas. Although the technique varied depending upon the portion of the esophagus to be resected, the principles of using the colon for replacement remained the same in all instances.

Benign and malignant lesions in the cervical region are operated upon with the patient in the supine position. If a thoracotomy is deemed necessary, the right side of the chest is elevated to a 45-degree angle. The esophagus is exposed in the neck through an incision along the anterior border of the right sternocleidomastoid muscle. The strap muscles are divided transversely over the thyroid gland and the latter retracted medially. The esophagus is isolated just above the manubruim and dissected upward, care being taken to identify and preserve the recurrent laryngeal nerve.

Concomitantly the abdomen is opened through an upper midline incision from the xiphoid process to well below the umbilicus. The lesser peritoneal cavity is entered midway along the greater curvature to the stomach and the gastrocolic omentum divided to the hepatic flexure. The lateral avascular peritoneal attachments of the ascending colon and cecum are severed and the entire right colon reflected medially. The appendix is removed whenever the cecum is to be used for the esophageal anastomosis. The middle, right, and iliocolic arteries are identified. The right colic artery is occluded close to its origin with a bulldog clamp. This allows observation of the adequacy of the middle colic artery to supply the cecum through the marginal arteries. The gastrocolic omentum is divided distally to the splenic flexure while the right colic vessel is occluded so that no operating time is wasted. If the circulation to the cecum appears to be adequate, the right colic and a branch of the iliocolic arteries are ligated near their origins and the ileum transected at the iliocolic junction. Following closure of the opening in the cecum, the right half of the colon is placed on the anterior chest wall to determine its adequacy for esophageal reconstruction. The mesentery on this side is divided down to the origin of the middle colic vessels. A tunnel is created in the anterior mediastinum just posterior to the periosteum of the sternum from the xiphoid process to the manubrium by blunt dissection from below and above (Fig. 3). The proximal end of the colon is passed into the neck through this tunnel using a long clamp inserted from above and down to the abdomen. To widen the thoracic inlet, the upper sternum has been split in four operations. Having determined that the cecum will adequately reach the neck without compromising its blood supply, the colon segment is isolated by dividing the transverse colon. Its mesentery is separated down to the middle colic vessels. Thus, the entire segment of the bowel is suspended upon a vascular pedicle comprised of only the middle colic artery and vein. The esophagus is transected above the lesion. The cervical esophageal colon anastomosis is performed end-to-end by the method of Sweet[21] with two or three layers of interrupted fine silk (Fig. 4). If the distal esophagus is not resected at this time, a catheter is inserted into this section and brought out through a stab wound to create a mucus fistula. Simultaneously, an opening is made on the anterior wall of the stomach and the distal end of the colon anastomosed to this organ. Either a pyloromyotomy or pyloroplasty may be performed. If a tube gastrostomy for preoperative feeding purpose has not

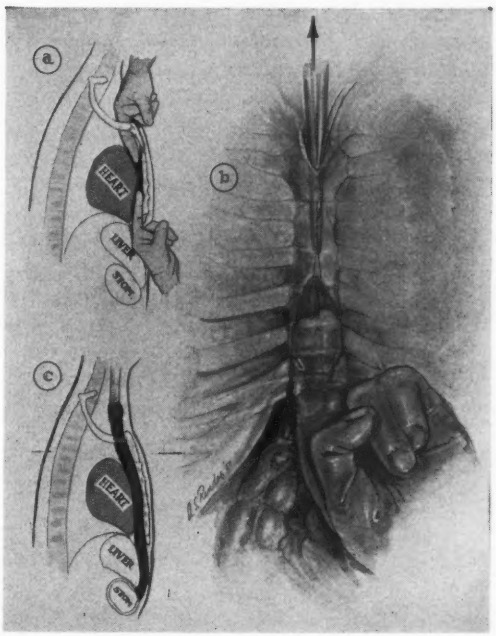


Fig. 3.—Drawing of anterior mediastinal tunnel created by blunt dissection from xiphoid to the manubrium.

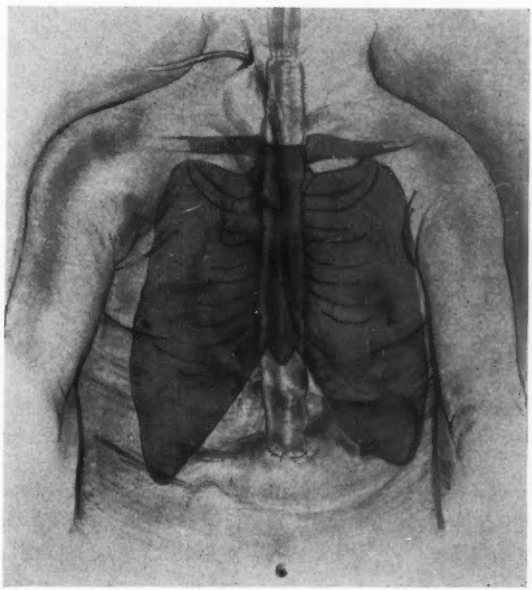


Fig. 4.—Drawing depicting completed anterior mediastinal colon transplant.

been done, this is placed to decompress the stomach in the early postoperative period. It may then be used for subsequent feedings until such time as the patient is able to eat normally. This maneuver obviates the necessity for an indwelling nasogastric tube. The continuity of the lower gastrointestinal tract is established with an end-to-end iliocolostomy.

Intrathoracic esophageal lesions above the aortic arch are approached through a right thoracotomy and upper right paramedian incision. The patient is positioned on the operating room table at a 45-degree angle. This allows the thoracic team to perform an adequate resection of the esophagus and invert the lower end. Simultaneously, with the patient in this position, the colon can be mobilized by the abdominal surgeon, if he will perform the operation while standing on the left side of the patient. Here the colon is delivered into the chest through an opening in the diaphragm anterior to the right lobe of the liver, and is placed in front of the hilum of the lung. The remainder of the operating technique is similar to that described previously (Fig. 5).

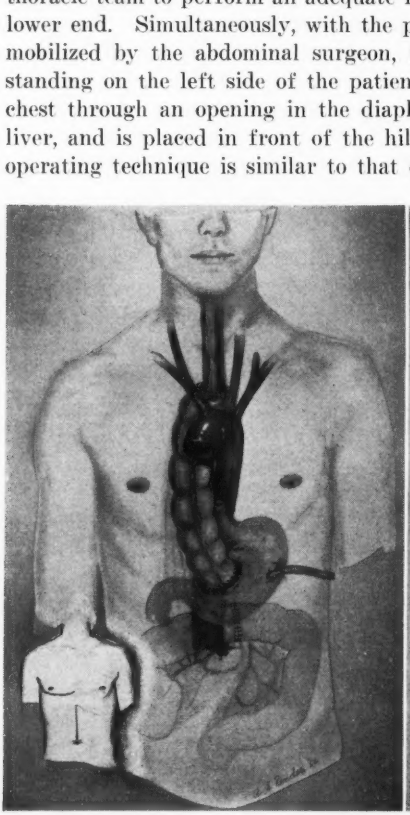


Fig. 5.

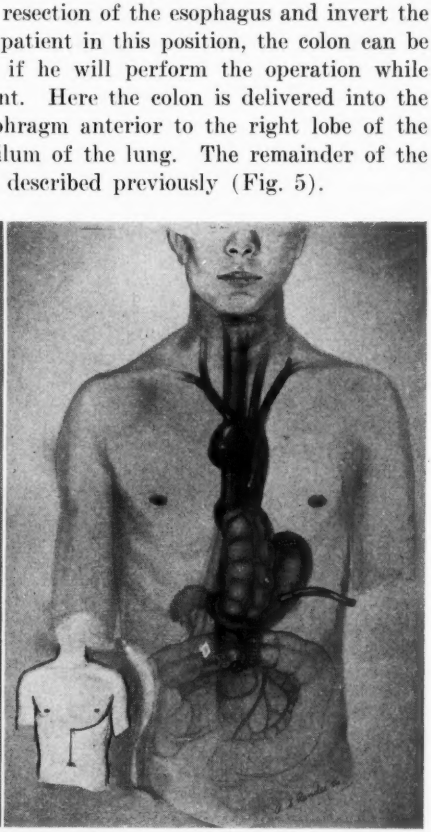


Fig. 6.

Fig. 5.—Drawing of completed intrathoracic colon transplant for lesions above the aortic arch.

Fig. 6.—Drawing of procedure for establishing upper gastrointestinal continuity following resection of intrathoracic lesions below the aortic arch.

Intrathoracic esophageal lesions below the aortic arch are approached through separate left abdominal and thoracic incisions. It has not been necessary to cut across the costal margin. The diaphragm can be transected from the hiatus outward to allow ample room for resection of the distal esophagus,

and cardia of the stomach if necessary. In this situation it may not be necessary to mobilize the entire right colon. The transverse colon is usually of sufficient length to substitute for the resected esophagus. Following an adequate resection of the lesion, the proximal portion of the bowel is passed into the left chest. The isoperistaltic esophagocolon anastomosis is performed in the manner previously described. The bowel is replaced in the bed of the resected esophagus and the entire area repleuralized. If the stomach is normal, the opening in the cardia is closed from above and the distal end of the colon is anastomosed to the stomach anteriorly. However, if a resection of a portion of the stomach is necessary, the distal end of the colonic segment may be anastomosed end-to-end to the remaining portion of the stomach, a pyloroplasty and a tube gastrostomy are performed. A colocolostomy completes the operative procedure (Fig. 6). The average operating time on all of the patients was 3½ hours. Culture of the colon is done at the time of surgery to perform drug sensitivity studies, and the appropriate antibiotic is given postoperatively. The gastrostomy tube is placed on continuous suction for a period of 3 or 4 days. This method of gastric decompression makes the use of an indwelling nasogastric tube unnecessary. The patient is given nothing by mouth for at least 10 days. He is fed through the gastrostomy tube beginning on the sixth postoperative day. Oral fluids are allowed on the tenth postoperative day. The diet is gradually increased until the individual is eating in a normal manner.

RESULTS AND COMPLICATIONS

To date, the colon has been used to reconstruct the esophagus in 28 patients. As depicted in Table I, 23 had carcinoma, and the remaining 5 benign disease. The 7 deaths in the immediate postoperative period were in patients with carcinoma above the aortic arch. Five additional patients have died in the interim from a progression of their disease or coronary occlusion. At the moment, 16 patients are living.

TABLE I. RESULTS OF ESOPHAGEAL REPLACEMENT WITH COLON—MORTALITY

LESION AND LOCATION	NUMBER	DEATHS			LIVING	
		IMMEDIATE POSTOPERATIVE	LATE	AVERAGE SURVIVAL TIME (MONTHS)	ALIVE	SURVIVAL TIME (MONTHS)
Carcinoma above arch	12	7	5	8.2 (2-18)	-	-
Carcinoma below arch	11	0	0	-	11	3-36
Stricture (corrosive) total esophagus	1	0	0	-	1	5
Stricture (corrosive, atresia and esophagitis) below arch	4	0	0	-	4	4-21

One patient died of generalized peritonitis on the sixth postoperative day due to dehiscence of his cologastrostomy. This may have been related to gastric distention which resulted from failure to perform a pyloroplasty and a tube gastrostomy.

Two other deaths were preventable. One elderly man died during the night of a spontaneous pneumothorax. Another patient died following disruption of the cervical and thoracic incisions. By mistake, the sutures were removed on the sixth day, post-mortem examination disclosed leakage of gastric contents into the posterior mediastinum from a distended lower esophageal stump. Here again, a tube gastrostomy to decompress the stomach had been omitted.

At the outset of this present study, it was our feeling that in nonresectable carcinomas the patient might be given palliation if the lesion was bypassed with the colon. Upon establishing a satisfactory swallowing mechanism, the primary lesion could be irradiated. This was done in 4 patients with extensive high carcinomas. None of these patients left the hospital, and in none was roentgen therapy instituted. Even though they obtained an adequate caloric intake through the gastrostomy tube, wound healing was poor. Ultimate breakdown of one or more of the anastomoses occurred with a fatal outcome in each of these patients.

Five individuals died 2 to 18 months postoperatively due to metastasis or heart disease. All of these patients could swallow in a normal manner and initially gained weight. At the moment, 16 patients are living who have been followed from 3 to 36 months. One hundred per cent of the patients had a normal appetite with normal ingestion of food. Ninety-four per cent had an improved nutritional status and have gained weight. Hemoglobin, hematocrit, and serum proteins were normal in all individuals at one time in the postoperative period.

The commonest nonfatal complication was diarrhea. This occurred in half of the surviving patients and in all of the experimental animals. It is associated with ingestion of food. Fortunately, this usually persisted for only a few weeks.

Three of the earlier cases had varying degrees of gastric retention. The use of a pyloromyotomy or a pyloroplasty in association with suction through a gastrostomy has prevented this occurrence.

Empyema without a demonstrable anastomotic leak occurred in 2 patients. They responded satisfactorily to frequent early aspiration and subsequent open drainage.

In 3 patients, because the middle colic vessels arose from superior mesenteric artery and vein to the extreme right of the midline, it was necessary to use the distal end of the bowel for the esophageal anastomosis. All of these patients complain of nocturnal regurgitation. Reverse peristalsis can be demonstrated in these segments fluoroscopically.

Atelectasis has been a disturbing complication in resecting high intrathoracic carcinomas. In 4 elderly patients, collapse of the right lung occurred soon after surgery. All responded satisfactorily to bronchoscopy and tracheostomy.

Narrowing of the esophago-colon anastomosis in the neck occurred in 2 patients. One followed closure of a fistula and the other was due to angulation. Revision was successfully carried out in both instances.

CASE REPORTS

CASE 1.—*Stricture of the entire esophagus* (Fig. 7): B. S., a 30-month-old Negro male child, was admitted to Cleveland City Hospital on Nov. 20, 1956, because of persistent regurgitation of food of 2 weeks' duration. No history of ingestion of corrosive material could be obtained from the mother. Barium swallow on Nov. 23, 1956, revealed two areas of narrowing of the esophagus, one at the midportion and the other 2 cm. proximal to the cardia. Esophagoscopy on Nov. 27, 1956, demonstrated narrowing of the lumen at 13 cm. The mucosa appeared reddened and friable. No attempt was made to pass the esophagoscope beyond this level, but biopsy was obtained which subsequently proved to be noncontributory. Several hours following esophagoscopy, the baby developed crepitation of the neck. A roentgenogram of the chest revealed subcutaneous emphysema and a pneumomediastinum. He was immediately taken to surgery where a superior mediastinotomy and a tube feeding gastrostomy was performed. Aside from the subsequent single aspiration of 120 c.c. of straw-colored fluid from the left hemithorax, his recovery was uneventful. Progress x-ray studies showed narrowing of the esophagus, and because of the unknown etiology, a secondary esophagoscopy was undertaken on Jan. 14, 1957, which demonstrated a white scarred ring 11 cm. from the incisor teeth. A No. 5 esophagoscope could not be passed beyond the stricture. No attempt was made to biopsy the area because of the previous unfortunate

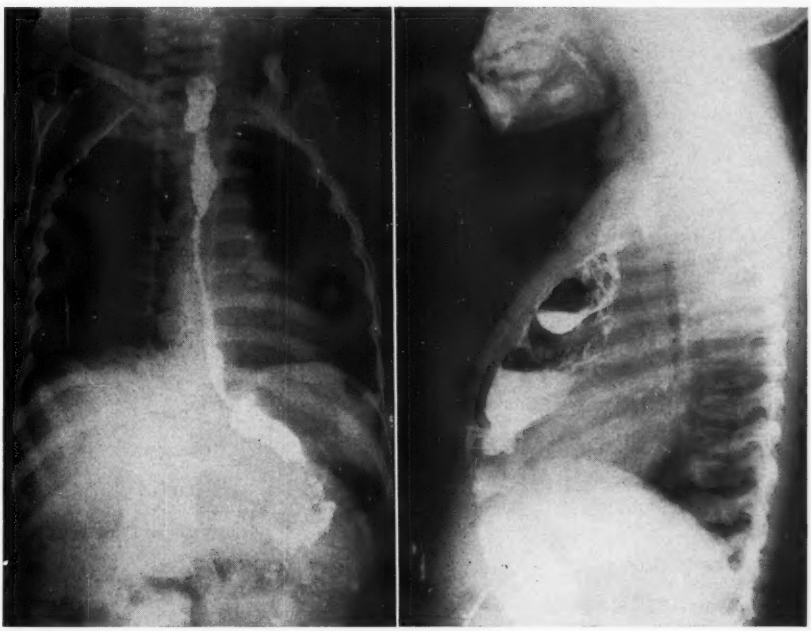


Fig. 7.—Roentgenogram on right shows a long esophageal stricture. Postoperative film 5 months following colon transplant through anterior mediastinal tunnel.

episode. The child was prepared for surgery in the usual manner and operated upon Jan. 30, 1957. Because the stricture began in the cervical region, we deemed it advisable to perform a two-stage procedure upon this child, primarily to interpolate the colon between the normal esophagus in the neck and the stomach through a mediastinal tunnel without opening the chest, and subsequently to resect the entire remaining esophagus via a right thoracotomy. The operation was performed with the child in the supine position. The esophagus was exposed in the neck through an incision along the anterior border of the

right sternocleidomastoid muscles. A V-shape segment of the sternum was removed to increase the anterior posterior diameter at the thoracic inlet. This maneuver allows the colon to be displaced into the neck without constriction at this level. The esophagus was transected at the level of the manubrium and a small catheter inserted into the distal end in an attempt to create an external mucus fistula. This was brought out through a stab wound lateral to the original incision. Through a separate abdominal incision, the colon was mobilized from the cecum to the splenic flexure in the manner previously described. A tunnel was created in the anterior mediastinum from below and above. The cecum was displaced into the neck through this tunnel and end-to-end esophagocolostomy was performed 2 cm. below the inferior pharyngeal constrictor muscles. The distal cut end of the colonic segment was anastomosed to the stomach and end-to-end iliocolostomy established lower gastrointestinal continuity. The pyloroplasty completed the operation.

His postoperative course was most gratifying. Gastrostomy feedings were begun on the seventh day and oral feedings on the eleventh. He was discharged on February 20, at which time he was eating chopped foods normally.

He was readmitted to the hospital on March 26. The mother stated that he had experienced some difficulty in swallowing in the previous 2 weeks. X-ray studies of the esophagus and transplanted colonic segment demonstrated narrowing of the esophagocolostomy. The barium however easily traversed this area into the stomach. Hemoglobin, hematocrit, and serum proteins were normal.

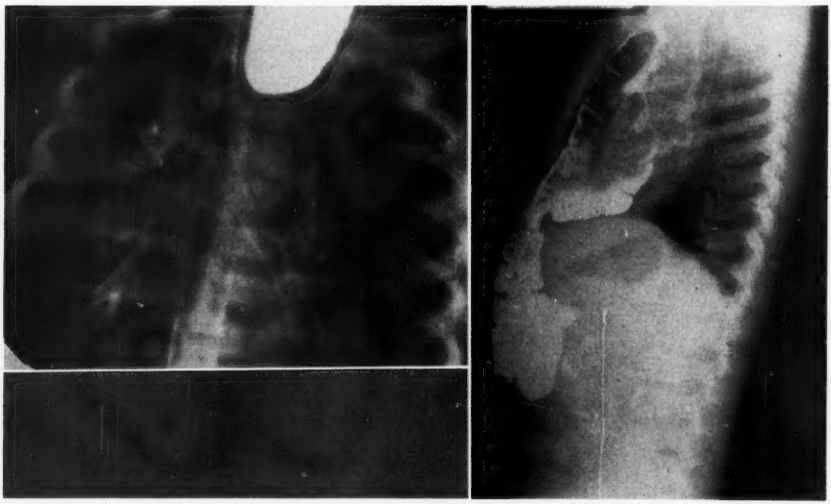


Fig. 8.—Delayed reconstruction of esophagus for tracheosophageal fistula with atresia. Anterior mediastinal colon transplant 21 months postoperatively.

CASE 2.—*Delayed reconstruction of esophagus in tracheoesophageal fistula with atresia* (Fig. 8): Baby F. was operated upon July 22, 1954, at the age of 4 days for a tracheoesophageal fistula with atresia. After ligation of the fistula, the lower end of the esophagus was resected. A feeding gastrostomy and cervical esophagostomy were performed.

Due to the extreme diligence in the care of this child at the age of one year he was felt to be a suitable candidate for a reconstruction of the esophagus. From previous experience in the use of the colon for esophageal replacement following resections for carcinoma, we felt that this segment of bowel could be used for almost a total esophagoplasty.

He was operated upon Aug. 27, 1955. With the baby in the supine position the abdomen was opened through a long upper midline abdominal incision. The colon was mobilized

from the cecum to the splenic flexure. It was transected proximally and distally after an adequate length for esophageal reconstruction had been determined.

The cervical esophagostomy was dissected free and a tunnel created in the anterior mediastinum just posterior to the periosteum of the sternum. The proximal end of the colon segment, which was suspended on a pedicle of the middle colic artery and vein, was passed through the mediastinal tunnel into the neck. An esophagocolostomy and cologastrostomy were performed concomitantly. A colocolostomy reunited the lower bowel.

His postoperative course was uneventful except for the development of a cervical fistula. This healed readily and he was eating normally at the time of his discharge.

On Dec. 15, 1955, he was admitted to the hospital because of difficulty in swallowing. It was obvious that he had a stricture at the esophagocolostomy but, because of severe diarrhea and an elevated temperature, a revision of his anastomosis was postponed. On December 18, he suddenly became cyanotic and dyspneic necessitating an emergency exploration of the neck. Although it was our intention to perform an immediate tracheostomy, the dilated upper esophageal segment, which was adherent to the skin, was opened. Marked narrowing of the colon was found at the thoracic inlet. The stricture was manually dilated with a clamp until an adequate opening was obtained. The esophagus was closed and a tracheotomy performed. He made a satisfactory recovery and today is a healthy active child.

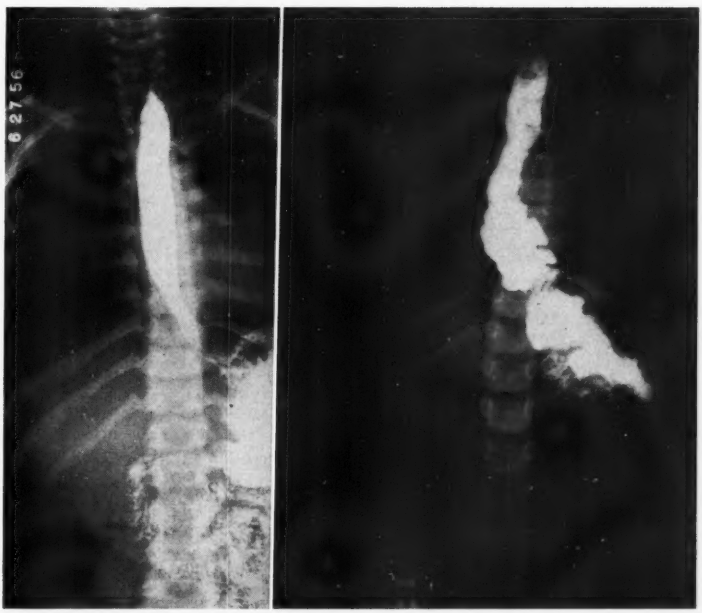


Fig. 9.—Colon transplant for lower esophageal stricture following ingestion of muriatic acid.

CASE 3.—*Stricture of the lower esophagus following ingestion of corrosives* (Fig. 9): J. S., a 6-year-old boy, was admitted to University Hospitals on Oct. 19, 1953, shortly after swallowing muriatic acid. An emergency tracheostomy was performed because of difficulty in breathing. He was placed on cortisone and antibiotics. Barium swallow one week after admission demonstrated a stricture at the lower end of the esophagus and pyloric obstruction. Bouginage of the esophagus was successfully carried out but the pyloric obstruction persisted. Resection of the distal end of the stomach was done on Dec. 10, 1953, a Bilroth I type of procedure being performed. Despite repeated esophageal dilatations, dysphagia and

vomiting persisted. Neuropsychiatric therapy was instituted because of absence of roentgenographic evidence of pyloric obstruction. Narrowing of the distal end of the esophagus was repeatedly evident by x-ray study and esophagoscopy. On several occasions it was necessary to remove a bolus of food from the esophagus. In June, 1955, x-ray studies of the esophagus and stomach showed 90 per cent gastric retention at the end of 6 hours. He was admitted to St. Alexis Hospital where revision of the gastroduodenostomy was done on July 1, 1955. The lower end of the esophagus was definitely narrowed but there appeared to be normal passage of barium through this area. Following this he was relieved of his symptoms only temporarily. He was admitted to City Hospital on July 7, 1956, for consideration of resection of the esophageal stricture. Roentgenograms showed a persistent narrowing at the lower third of the esophagus with an adequate gastroduodenal stoma. On July 12, 1956, the lower end of the esophagus was resected and the transverse colon interpolated in an isoperistaltic manner between the esophagus and the stomach. His postoperative course was uneventful except for transient rise in fever in his second postoperative day. Blood culture was done at this time and it was positive for *Aerobacteraerogenes.* Tube feedings were begun on the seventh postoperative day and oral feedings on the tenth day. He was discharged on the sixteenth postoperative day at which time he was eating a normal diet. The gastrostomy tube was removed just prior to his leaving the hospital. He was readmitted to the hospital on March 12, 1957, for follow-up studies. In the interim his mother stated that he was now able to ~~eat~~ everything and had no regurgitation. His only complaint was occasional abdominal cramps and diarrhea. Weight gain had been approximately 4 pounds in 2 months. X-ray studies showed slight delay in the passage of barium from the esophagus into the colonic segments but was otherwise normal. Blood studies performed at this time showed a hemoglobin of 12 Gm., white blood count of 6,780. Total proteins were 6.4, albumin 4.9, globulin 1.5 with an A/G ratio of 3.27.

CASE 4.—*Carcinoma of the distal end of the esophagus with invasion of the stomach* (Fig. 10): R. W., a 49-year-old Negro man, was admitted to Cleveland City Hospital on May 20, 1956, complaining of dysphagia of one year's duration. During the interim he had lost 40 pounds of weight. Esophagrams demonstrated an irregularity of the distal third of the esophagus with almost complete obstruction at this level. On esophagoscopy, a squamous-cell carcinoma was biopsied 40 cm. from the incisor teeth. Following satisfactory chemical and nutritional balance and the usual bowel preparation, the patient was operated upon Jan. 11, 1956. With the patient in the lateral position, the left chest was entered through the seventh interspace. The lesion was found to extend from a position approximately 9 cm. above the diaphragm down to and through the hiatus. The esophagus was mobilized from the aortic arch down to the diaphragm. The latter was incised anteriorly to the costal margin. Simultaneously through a left paramedium incision, the transverse colon was mobilized by the abdominal team. Since the lesion was found to be invading the stomach, the proximal two thirds of this organ was removed along with the lower esophagus. A section of the transverse colon on a pedicle comprised of only the middle colic vessels was brought up into the chest. The proximal end of the bowel was anastomosed to the esophagus about 4 cm. below the arch of the aorta. The distal end of the colonic segment was anastomosed end-to-end to the remaining portion of the stomach. A pyloromyotomy was done as was also a tube gastrostomy. Lower intestinal continuity was re-established with an end-to-end colocolostomy. Before closure of the chest, the segment of the colon in the thorax along with a proximal portion of the esophagus was entirely repleuralized. The duration of the operation was 4 hours, during which time he received 8 units of blood. Following extubation, he developed difficulty in breathing and it became obvious that the left lung was collapsed. A tube was inserted into the thorax anteriorly in the third interspace and, because of the tracheobronchial secretions, a tracheostomy was done. He responded satisfactorily to these measures and subsequently had an uneventful convalescence.

Pathologic sections demonstrated an extensive squamous-cell carcinoma of the lower end of the esophagus with invasion of the proximal portion of the stomach. The esophagus had been divided through normal tissue 2 cm. proximal to the lesion and the stomach divided 7 cm. distal to the lesion. The lymph nodes removed with the specimen were negative for

neoplasm. He was discharged on the thirty-fifth postoperative day. Since leaving the hospital, he has gained 45 pounds and is back to work as a laborer in the steel mill. His appetite is normal and he has no abdominal distress and no regurgitation. X-ray studies reveal normal passage of barium through the upper gastrointestinal tract. Hemoglobin, hematocrit, and serum proteins are within normal limits.

Fig. 10.—Carcinoma lower end of esophagus resected and colon transplant performed one year previously. Patient gained 45 pounds and works in a steel mill.

CASE 5.—*Benign stricture of lower third of the esophagus due to esophagitis and hiatal hernia* (Fig. 11): H. K., a 55-year-old white man, was first seen in Polyclinic Hospital in February, 1956, because of hematemesis and tarry stools of 3 days' duration. X-ray studies of the upper gastrointestinal tract were interpreted as showing evidence of esophagitis, and hiatal hernia with a 2 cm. constriction above the left leaf of the diaphragm. Esophagoscopy showed a concentric narrowing of the lumen 29 cm. from the incisor teeth. A biopsy of this area was reported as chronic esophagitis. In view of the symptoms, he was operated upon Dec. 4, 1956. The lower end of the esophagus was hypertrophied and one could feel a firm indurated area just above the cardioesophageal junction. Due to the shortened esophagus, about 4 inches of the cardia was above the diaphragm. The stomach was opened longitudinally just below the cardioesophageal junction and the incision continued up into the esophagus through the stricture. A frozen section of this segment disclosed no evidence of neoplasm. In view of this, the esophagus was closed transversely. The diaphragm was incised from the hiatus outward. A Foley catheter was inserted in the stomach and brought out through a stab wound in the left upper quadrant for decompression of the stomach in the early postoperative period. Because of the short esophagus, it was impossible to place the cardioesophageal junction below the level of the diaphragm. The postoperative course was uneventful. He was discharged December 19, eating a normal diet. X-ray studies of the esophagus and stomach just prior to discharge showed very little change in the pathologic entity. It was felt that an inadequate operation had been performed and that sooner or later this man would again get into difficulty and a different type of operative approach would be considered. He was readmitted to the hospital just 6 days later on December 24,

because of gastrointestinal bleeding. The hemoglobin was 2.6 Gm. with a hematocrit of 7. The treatment consisted of Sippy diet and multiple transfusions. He was discharged markedly improved on Jan. 18, 1957, with a hemoglobin of 11 Gm. He was admitted to the hospital a third time on March 12, 1957, complaining of persistent lower thoracic and epigastric distress. The hemoglobin was 8 Gm. and he had a hematocrit of 24. Tarry stools had been present for 3 days.

In view of the symptoms of recurring upper gastrointestinal bleeding, it was felt that the lower esophagus should be resected, the stomach placed beneath the diaphragm, and the esophagus reconstructed with a segment of the transverse colon.

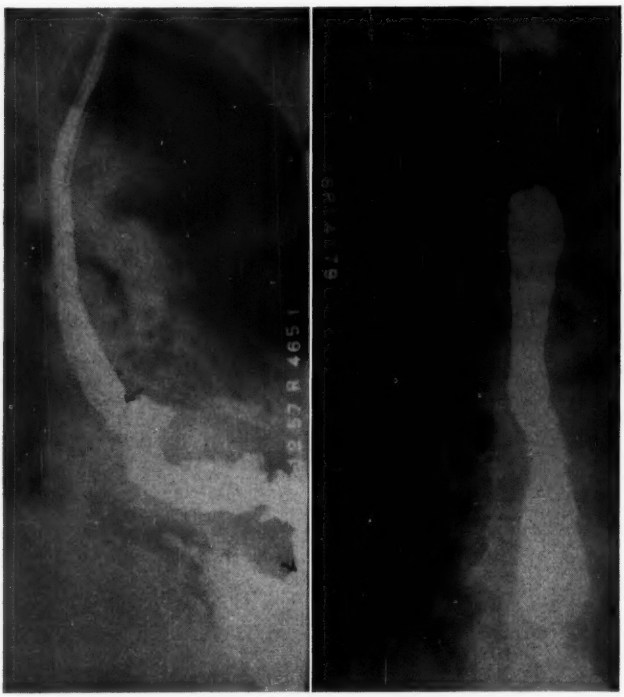


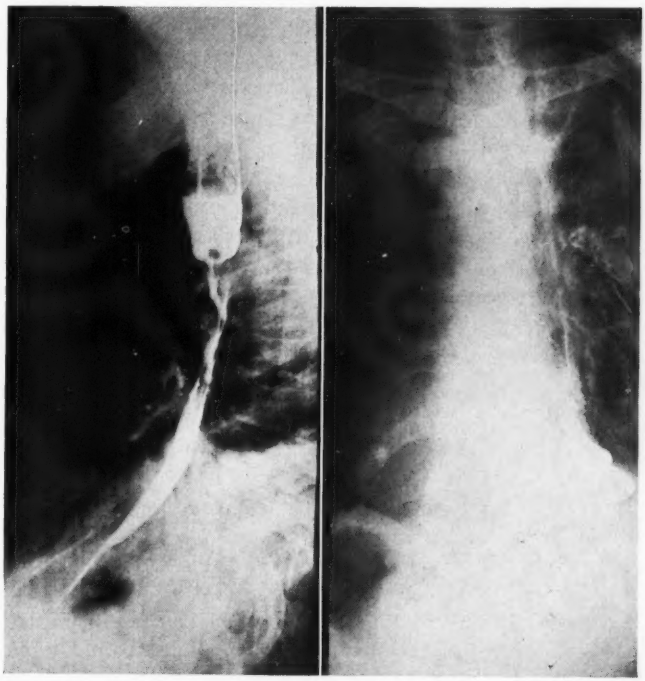
Fig. 11.—Esophagus reconstructed with the colon following resection of a stricture of lower end due to esophagitis and hiatal hernia. Previous Heinicke-Mikulicz procedure with a hiatal herniorrhaphy was unsuccessful.

This operative procedure was done on March 19, 1957. The abdomen and the left thorax were simultaneously opened. The lower end of the esophagus was resected and the cardia closed and isoperistaltic segment of transverse colon was interpolated between the esophagus and a separate opening at the fundus of the stomach. A colocolostomy, pyloroplasty, and a tube gastrostomy were performed.

The postoperative course was most gratifying. Gastrostomy feedings were begun on the sixth day and oral feedings on the tenth. On discharge from the hospital, he was eating satisfactorily and completely relieved of his symptoms.

CASE 6.—*Carcinoma above the aortic arch* (Fig. 12): D. S., a 53-year-old Negro man, was admitted to Cleveland City Hospital on April 8, 1956, with a left lower lobe pneumonia. He stated that for 3 weeks he had had difficulty in swallowing solids. An esophagram demonstrated a lesion at the level of the aortic arch. On esophagoscopy, a squamous-cell carcinoma was biopsied 25 cm. from the incisor teeth.

He was operated upon April 20, 1956. With the patient positioned on the operating table at a 45-degree angle, the right thorax and abdomen were simultaneously opened. The esophagus was resected by the thoracic team from 4 inches above the azygos vein down to the diaphragm and the lower end inverted. A frozen section of the upper end of the esophagus was negative for carcinoma. At the same time the colon was mobilized from the cecum to the splenic flexure. It was severed proximally and distally and the cecum delivered into the chest through the diaphragm anterior to the liver. An esophagocolostomy and colo-gastrostomy established upper gastrointestinal continuity. An iliocolostomy, pyloroplasty, and tube gastrostomy completed the operative procedure.



A. *B.*

Fig. 12.—Esophagus reconstituted with the colon following resection of carcinoma above the arch.

A, Preoperative: Negro man, aged 54; carcinoma above aortic arch.

B, Postoperative: Resection with colon transplant anterior to right hilum. Patient living and well at 10 months.

His postoperative course was rather prolonged and stormy. He developed an incarcerated hernia which was repaired on the tenth day after operation. An empyema in the right thorax was drained on the sixteenth day. Following this, an esophagopleural cutaneous fistula was demonstrated. This healed in a short time and at the time of his discharge he was eating in a normal manner.

He subsequently gained weight and felt well until November, 1956. He returned to the out-patient clinic at this time complaining of difficulty in swallowing. Esophagoscopy revealed recurrent carcinoma near the esophagocolon anastomosis. (It is interesting that sections through this area at the time of surgery showed no evidence of neoplasm.) X-ray therapy was instituted with only temporary improvement. He died recently at another hospital. No post-mortem examination was performed.

DISCUSSION

Reconstruction of the esophagus has been a problem since the first successful resection in 1913 by Torek.[23] Initially antethoracic skin tubes enjoyed considerable popularity. However, the multiple stages with numerous failures due to strictures and fistulas caused this to be abandoned.[25] The stomach has been used subcutaneously and intrathoracically since the first successful reconstruction by Adams and Phemister in 1938,[1] following resection of a carcinoma of the lower third. Due to the work of Sweet[21] and Garlock[5] higher and higher transplantations have been performed. The stomach has distinct advantages—it is readily available, has a good blood supply, and can be mobilized easily. However, the development of peptic esophagitis, regurgitation of gastric contents into the pharynx in high resections, and the general unhappy state of many of these individuals in regard to their eating habits are distinct disadvantages. The development of esophagitis and gastritis in our experimental animals within a relatively short period of time lends support to the thesis that the stomach is not a good substitute for the esophagus.

Subcutaneous jejunal transplantation was first used successfully by Roux in 1907.[19] In recent years, intrathoracic jejunal replacement of the esophagus has received considerable impetus from the work of Rienhoff,[16] Harrison,[8] and Robertson.[17] However, the short vascular arcades make it difficult to obtain a long healthy segment.

Although inert plastic tubes could serve as an esophageal bridge, from our own personal experiences in the laboratory,[13] we do not believe this should be done in man.

The use of the colon to replace the esophagus is not a new idea. It was used sporadically in Europe as an antethoracic subcutaneous transplant since 1911. However, very little has been written concerning the intrathoracic replacement of the esophagus with the colon. Robertson, Howe, and Smithwick[17] reported, before the Surgical Forum in 1952, a successfully staged operation in which the transverse colon was interposed between the esophagus and jejunum. Kergin, in 1953,[10] used an isolated transverse colonic segment to bypass a high esophageal obstruction caused by a benign paraffinoma. In 1954, Mahoney and Sherman,[11] and Dale and Sherman[4] reported upon their experiences using the right colon for reconstructing the esophagus in carcinoma and tracheoesophageal fistula with atresia. Recently Haight[7] has successfully used the right colon in 7 cases for delayed reconstruction of the esophagus in tracheoesophageal fistula with atresia.

In order satisfactorily to replace the esophagus, the substitute should meet certain criteria. It should have sufficient length, be assured of continuing viability and the lining be resistant to acid peptic digestion. The colon appears to meet these requirements and in addition offers other distinct advantages. The colon has an excellent blood supply. The anatomic distribution of the marginal arteries is such that a segment of viable bowel suspended upon a pedicle of the middle colic artery and vein can be mobilized from the cecum to the descending colon.

Recent evaluation of the poor results obtained in resection for carcinoma of the esophagus significantly point to the high incidence of local anastomotic recurrence due to inadequate longitudinal removal of the esophageal cancer.[20, 24] The colon is of sufficient length to reach the neck in high esophageal resections. This allows one to resect as much of the esophagus as deemed necessary and do an anastomosis without tension at the proximal suture line. Although the majority of our esophago-colon anastomoses have been performed within the thorax, it may be better to do them in the neck. Less proximal colon has to be used when the bowel is displaced through an anterior mediastinal tunnel into the neck because of relatively short distance between the xiphoid and the manubrium. The vascular pedicle is not angulated or put under tension as is occasionally true when the colon is displaced into the right chest over the right lobe of the liver. One great advantage of doing the anastomosis in the neck is that if leakage occurs the resultant fistula can easily be closed and will not be fatal.

We have been particularly impressed with the length of the colon in children. This is due to the redundancy of the transverse colon and the presence of a thin pliable mesentery. Thus, it is particularly applicable in reconstituting an adequate swallowing mechanism in congenital atresia or corrosive strictures where long segments are involved.

The large bowel is seemingly resistant to peptic ulceration clinically even though reflux of barium into the colonic segment can be demonstrated fluoroscopically, no evidence of colitis could be seen on routine esophagoscopy and post-mortem examination up to 20 months following implantation.

Our experimental evidence supports the above concept. Furthermore, Merendino and his associates[12, 22] subjected dogs to histamine stimulation 30 days after colon transplants. None of their animals exhibited esophagitis or colitis. However, in most of his animals, severe gastritis was present.

The absence of esophagitis clinically and the inability to produce it experimentally when the colon is interposed between the esophagus and stomach suggests its applicability in resections for peptic esophagitis. Although 2 patients had complete amelioration of their symptoms, their follow-up has been too short to propose this operative procedure as a cure in these individuals. With the evidence at the moment, it appears that the interposition of an acid resistant segment of bowel between the esophagus and stomach protects the usual susceptible esophageal muscosa from digestion by regurgitation of gastric juices. Also the use of a segment of bowel between the esophagus and the stomach allows the latter to remain in its normal position below the diaphragm.

Fluoroscopic examinations show regular contractions which propel the bolus along the transplanted colon to the stomach. Thus, the colon serves as a conduit between the esophagus and the stomach. This, along with the stomach remaining in a normal position below the diaphragm, accounts for the excellent nutrition of these patients.

SUMMARY

1. The technique of reconstruction of the esophagus with segments of the colon is outlined.

2. Experimental data are presented to support the thesis that the colon can serve satisfactorily as an esophageal substitute without the development of colitis or esophagitis.

3. The results and complications in 28 patients operated upon for benign and malignant disease are discussed.

4. The advantages of using the colon instead of the stomach or jejunum are presented.

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Discussion

DR. EARLE B. MAHONEY, Rochester, N. Y.—Thank you, Dr. Haight, for the privilege of discussing this paper. Dr. Sherman, Dr. Dale, and I have been extremely interested in following Dr. Neville's work and we are delighted with the results he has had, with his present enthusiasm for the procedure, and the beautiful presentation given today. Our series is not as large as Dr. Neville's but I think we agree in every way with what he has said. We are particularly interested in using this as a procedure for establishing continuity of the gastrointestinal tract in children with benign lesions. The original procedures were for carcinoma of the upper esophagus and tracheoesophageal fistulas in which we could not establish normal continuity by direct anastomosis. Those children are now growing and are well at 3 years of age. We also feel that in the case of carcinoma of the esophagus, as mentioned by Dr. Neville, that if one can do a total esophagectomy the end results will be better. We have used this several times as a two-stage procedure in poor-risk patients, resecting the carcinoma as the first stage through a thoracic incision on the right, leaving a cervical esophagostomy and a gastrostomy. As soon as the patient's general condition improves the gastrointestinal continuity is restored by a colonic transplant in the anterior mediastinum which does not require a second thoracotomy.

DR. FRED JARVIS, Seattle, Wash.—Although we too have used the colon in replacement of the esophagus, I should like to present a somewhat varied situation, which we encountered recently, with the thought that its solution might be of value to others in the Association.

(Slide) This lady, aged 54 years, presented herself with a markedly decompensated achalasia of the esophagus that held 1,300 c.c. In 1948, we did the then accepted cardioplasty procedure. The result was only moderately successful as we had found in a series of 9 cases at that time, and we classed her as a fair result only. However, her symptoms continued mildly for approximately 6 years and then more severely for a year because of reflux esophagitis, until she finally was unable to get anything through the strictured lower end of the esophagus, even liquids. We then chose to interpose a segment of jejunum about 25 cm. in length, this operation having been applied in 2 cases of achalasia at the time, very successfully I believe, by Merendino and Dillard. The result again was only moderately and briefly successful, because at the end of 6 weeks she was again totally unable to eat.

(Slide) This was the stricture that existed at the time she returned.

Six weeks after the interposition of the jejunum we found a situation on esophagoscopy that was misinterpreted as a stricture of the proximal anastomosis of the jejunum. We chose then to enter the right chest since we could not get into the proximal stoma to insert a dilator. We found a most interesting situation. The large sigmoid esophagus had developed a cecumlike dependent pouch beyond the jejunal stoma. The anastomosis was widely opened proximally. Two fingers could be inserted into the stomach. Weight of food in the esophageal pouch converted the proximal stoma into a slitlike aperture that was physiologic rather than organic. The problem of dealing with this became one that we puzzled about for a bit. Finally, at the suggestion of my associate, Dr. Robert Coe, we decided that if we could convert this very large sigmoid pouch into a straight, though aperistaltic, tube to deliver the food into the peristaltic jejunal hopper below, it might work. We therefore made a wide V excision of the excess esophagus from the thoracic inlet down to the cecum and thus recreated a small, relatively normal-sized esophagus.

The result has been most satisfactory. The patient now for the first time, since she was 18 years old, is able to eat steak and anything else she wants as rapidly as anyone.

(Slide) Unfortunately, this slide shows quite poorly the final result. There is very slight hesitancy of barium at the orifice of the interposed segment. Barium passes otherwise normally and rapidly into the stomach.

DR. GEORGE I. THOMAS, Seattle, Wash.—As you know, we have been interested in the replacement of the esophagus and portions thereof by the jejunal interposition operation, for approximately 5 years, under Dr. K. A. Merendino's guidance, and I thought it would be of interest to the Association to bring up to date some of our recent data.

(Slide) This slide was alluded to by Dr. Neville and represents a comparative study of groups of jejunal and colon replacement preparations. Fourteen dogs in the colon series and 10 dogs in the jejunal series were given large doses of histamine for intense stimulation of acid-pepsin secretion and evaluated either upon their death or upon sacrifice after 45 days. As you can see, both segments of the intestinal tract presented esophagitis grossly and microscopically. The colon and the jejunum essentially had the same negligible degree of pathology after this stimulation occurred. One perforation of the colon is included at the gastrocolic anastomosis and, at the time of autopsy, it contained a small amount of inspissated pus, and we therefore perhaps assumed that it was due to a suture line breakdown. You can see a similarity of ulcerative disease of the stomach and duodenum in both series.

(Slide) This is a recent breakdown of 29 clinical cases. The jejunal segments vary in length from 20 to 37 cm., the majority of which (18) were done for benign peptic esophagitis with stricture. All but 2 of these patients had sliding hiatal hernia. We have performed 3 jejunal interposition operations for cardiospasm, and 3 operations for this entity following several previous procedures, esophagogastrostomy and similar operations. We have performed 2 procedures for extrahepatic portal block with varices, and these patients are doing well. There are 3 miscellaneous lesions, 2 lye strictures, and one iatrogenic (?) stricture. That was a lady who developed a severe peptic esophagitis and stricture following prolonged nasogastric intubation after cholecystectomy. The mortality rate is 10 per cent in the entire series.

(Slide) We think this slide will be of interest, because of the fact that all these patients were severe alcoholics. We believe that alcohol is much like histamine in the experimental preparation and puts the intestinal "esophagus" to severe test. As a matter of fact, 3 of these patients had suffered because of their stricture, and were able to resume their very pleasant pastime following the jejunal procedure. They are very happy with the operation.

DR. OWEN H. WANGENSTEEN, Minneapolis, Minn.—One of the great advantages of having graduate students is that unanticipated dividends accrue therefrom not infrequently and, when these men come from other institutions, they often bring with them ideas concerning operations new to the recipient institution, but practices already in vogue from whence they came. The nature of my discussion will outline such an occurrence.

Many of you in this audience, I know, have worked with various types of gastric tubes for esophageal replacement. A number of you have used the Beck-Jianu gastric fundus. A number of years ago (Surg. Gynec. & Obst. 72: 257, 1941), I reported upon 5 patients in whom I had swung up a jejunal loop attaching the lower end to the gastric fundus, the upper proximal portion being brought up subcutaneously in the midline to the nipple level. This procedure was employed to complete the Torek operation for cancer of the esophagus. There was no mortality although I completed few of the procedures.

It is concerning use of the Mes greater curvature tube (J. Internat. Coll. Surg. 11: 270, 1948) in replacing the esophagus in operations for cancer of the upper thoracic segments of which I wish to speak today. Whereas the Beck-Jianu tube, pedicled at the base of the gastric fundus, does give considerable added length, it does not afford the length which a tube designed from the greater curvature does, if one sacrifices the entire lesser curvature of the stomach. Those of you who have measured the length of the greater curvature of the stomach at operation have had occasion to observe that it is in the range of 55 to 60 cm.; the lesser curvature from the pylorus to the esophagus, on the contrary, measures only usually from 20 to 25 cm. In other words, if one begins fashioning the greater curvature tube by cutting away the lesser curvature, beginning a short distance above the pyloric artery, it will be found that it is not difficult to make a tube about as long as a thread placed along the greater curvature from esophagus to pylorus. As has been suggested, this distance is ordinarily in excess of 50 cm. In making such a tube, I use the ordinary half-length, hysterectomy type of clamp, engaging the stomach along the path of the tube to be designed between two such clamps—the upper row of clamps will come away with the sacrificed lesser curvature. When this division has been completed, I remove the lower clamp and under the tension of stretch,

I place a series of interrupted sutures in the mucosa; the next clamp is removed and interrupted mucosal stitches are placed at this level, the surgeon taking pains to see to it that the tube is stretched out as it is being fashioned. When the mucosal suture has been completed to the very top of the gastric fundus, a similar row of interrupted sutures (4-0 silk) is placed in the serosa.

A Heineke-Mikulicz pyloroplasty is made to ensure ready gastric emptying. The length of the tube is measured upon the anterior chest wall to see that it will reach well into the neck. It is then brought up through the esophageal hiatus. I do these operations conventionally through the right thorax. A short transverse incision is made in the neck on the right side just above the clavicle. With two fingers of the left hand indenting and elevating the pleura, the neck wound is enlarged until the fingers from within the thorax protrude through the neck wound. The anterior portion of the sternocleidomastoid muscle is cut free from the clavicle, thus enlarging the wound and making it possible to pull the gastric tube through the esophageal hiatus behind the right lung into the neck.

I have done only one such operation. I did it in two stages, bringing the cervical esophagus out just beneath the cricoid cartilage beside the gastric tube. It could, possibly, have been done as a one-stage procedure. There seemed to be some question of the viability of the upper end of the gastric tube; however, as it proved, no slough in the gastric tube occurred. A catheter was left in the esophageal end for purposes of removing the swallowed saliva by suction. A four-hole No. 14 Fr. duodenal tube was placed in the retroperitoneal portion of the duodenum by the Witzel technique, its tip being threaded well into the gastric tube which lay within the thorax. During the early postoperative course, suction was applied to this tube to keep the gastric tube empty. A feeding jejunostomy was made also on the efferent loop of the jejunum just beyond the duodenojejunal angle.

Approximately 2 weeks after the primary procedure, union was established between the upper end of the gastric tube and the cervical esophagus by a single row of sutures. Disarticulation of the proximal end of the clavicle made this suture easy. On completion of the procedure, the capsule of the medial end of the clavicle was tacked down with a few sutures of interrupted chromic catgut. This patient has done well and has no trouble in swallowing. There was a temporary cervical fistula which closed quickly.

A number of surgeons have expressed deep disappointment and a sense of frustration over their accomplishment with cancer of the upper end of the esophagus. In his Judd Lecture at the Medical School of the University of Minnesota in February of this year, Mr. Philip Allison, distinguished Nuffield Professor of Surgery at Oxford University, England, indicated that he had given up operating for cancer of the upper thoracic esophagus because of the high mortality and the low cure rate. A number of distinguished American surgeons who have had a special interest in surgery of the esophagus also have sounded similar notes of discouragement over surgical removal of the proximal half of the thoracic esophagus for cancer.

(Addendum on proof reading discussion, July 3, 1957.—Since this discussion took place, the writer has twice swung up a Mes greater curvature gastric tube into the neck for cancer of the upper thoracic esophagus. In both instances, a one-stage anastomosis was made. Both concerned very difficult cancers, one of which was a frankly palliative procedure, in that the patient has involvement of the right lung and an esophagobronchial fistula between the esophagus and the right main bronchus. It was believed that division of the esophagus above the fixed cancer would mitigate the effects of the esophagobronchial communication. The lower end of the esophagus was anastomosed to a jejunal loop to provide a drainage vent for the esophageal secretions. This patient died a few weeks after operation from complications of the fistula. A small leak developed late in the neck wound, which did not affect the issue. Inasmuch as the cancer had been left in situ, the Mes tube had been brought up behind the sternum. In the other patient, even though the esophagus and trachea seemed to be fused into a single indurated mass, it was possible to separate them, working alternately from above and below. This patient has done well.)

In looking back upon this very limited experience with only 3 cases, the writer feels constrained to remark that the simpler convalescence of the patient with the two-stage procedure was noticeable. It was also the easier cancer to extirpate. At this time the writer feels that an added experience with both the one- and two-stage procedures will be necessary to determine which is the safer and better operation. Certain I am that this operative technique has something to offer in the management of cancer of the thoracic esophagus.

It is my impression that use of the Mes greater curvature gastric tube may go a long way to simplify some of the difficulties of esophagectomy for cancer of the upper half of the thoracic esophagus. Moreover, I believe that its use may also play an important role in improving the end results in that a greater length of the esophagus proximal to the lesion will be sacrificed in the extirpation of the cancer.

DR. RICHARD H. SWEET, Boston, Mass.—I would like to make one or two comments about the use of the colon. This is something we are all very much interested in nowadays, and it has great use. I believe with Dr. Mahoney that its utilization perhaps should be confined to 2 indications: (1) the use for substitution in cases of stricture and other benign lesions, particularly with regard to making an anastomosis in the neck, and (2) in cases of carcinoma for palliation only.

It is much easier to accomplish in children than it is in adults; at least that has been my experience. From the point of view of technique, so far as I am concerned, the exact methods of doing this are not quite established satisfactorily. I would like to point out to you that in cases of technical difficulty, which I shall not go into at the present time, I have found it useful to make a sternotomy incision all the way through the sternum. This can then be extended into the abdomen. This method has many advantages, one of which is that the surgeon does not have a second team that gets in his way.

One other comment is about the function or the physiologic behavior of this colon transplant with which we do not yet have much experience. I have one patient who is enormously satisfied with the operation because she is so much better than she was beforehand, but during defecation she has a most annoying fluttering sensation in her chest.

DR. WILLIAM E. NEVILLE, Cleveland, Ohio (Closing).—I wish to thank all the discussants. Dr. Mahoney was particularly kind. It was due to his initial enthusiasm using the colon as an esophageal substitute, that we embarked upon this program 3 years ago.

Dr. Jarvis mentioned using the jejunum and I believe we can go along with what he said. When we started this project it was our purpose to evaluate the colon only as an esophageal replacement. It may be better in lower esophageal lesions to use the jejunum. It is easier to obtain a segment of jejunum for a short lower esophageal replacement than to mobilize the colon.

We have been interested in following the work of Drs. Thomas and Merendino with regard to the interposition of a jejunal segment between the lower end of the esophagus and the stomach. Their results seem to be good. He did not mention whether they had used the colon clinically, but I know they operated upon a number of experimental animals using the colon and came to the conclusion that it has a certain inherent resistance to acid peptic digestion.

I thank Dr. Wangensteen for his comments. So far as using the stomach in the manner in which he depicted, even though it appears to be relatively easy to do—isn't this what we are trying to get away from in using the colon? With a tube of the stomach being used to replace the esophagus, you may end up with symptoms of esophagitis.

I agree heartily with Dr. Sweet. I disagree a bit on the point of palliation for carcinoma, if we can use that term. As I mentioned, we attempted to bypass 4 nonresectable carcinomas. In none were we ever able to institute roentgen therapy and none left the hospital. He is absolutely right in that it is much easier to mobilize the colon in a child. The colon seems to be longer, it has a thin pliable mesentery and you can get a much longer segment. The sternal incision is certainly an interesting approach. We have not used it because we have had no difficulty in obtaining an adequate anterior mediastinal tunnel.

I thank the Association for the privilege of the floor.

PRIMARY TUMORS OF THE HEART

A Surgical Problem

J. Gordon Scannell, M.D., and Hermes C. Grillo, M.D. (by invitation)
Boston, Mass.

PRIMARY tumors of the heart are clearly a surgical problem. As an increasing number of cardiac operations are carried out, a greater number of primary tumors of the heart will be recognized. Since the rapidly advancing techniques of cardiac surgery will certainly permit successful removal of many of these tumors, greater awareness of their clinical behavior and the employment of appropriate methods in their diagnosis are in order.

We wish to report the successful removal of two primary cardiac tumors. The first, a myxoma of the left atrium, was removed 22 months ago. The patient is now well. The second, a fibrosarcoma of the right atrium, was removed 12 months ago. This patient is presently well and without evidence of recurrence. A third patient, with an intra-atrial myxoma, died suddenly while awaiting diagnostic studies which almost certainly would have led to operation.

Case 1.[30]—The patient was a 33-year-old construction worker. He had been in excellent health until, 18 months before admission, he noted minimal hemoptysis during the course of a severe respiratory infection that was accompanied by an unusual degree of dyspnea. These symptoms subsided, but a second hemoptysis occurred 12 months later and again one month before admission. The dyspnea and wheezing associated with the latest episode were persistent and disabling. He therefore entered the hospital for study. There was no history of rheumatic fever.

On admission, the patient was dyspneic at rest. A few râles were noted at the right lung base, but there was no other evidence of congestive failure. Examination of the heart showed the classic signs of "pure" mitral stenosis. There was slight enlargement. The rhythm was regular. At the cardiac apex there was a presystolic roll of Grade 3 intensity, ending with crescendo in a sharply accentuated first sound. There was an associated presystolic thrill. The pulmonic second sound was moderately loud and there was an opening snap. No systolic murmurs were detected.

Electrocardiogram showed the pattern of right ventricular hypertrophy. X-ray study (Fig. 1) disclosed a slightly enlarged, triangular heart shadow with fullness of the left atrium and congestion of the lung roots.

Mitral commissurotomy was advised and operation carried out on May 23, 1955. The atrial appendage was of normal size and configuration, providing easy entry into the atrium. The latter was found to contain a soft lemon-sized mass attached by a narrow pedicle to the base of the interatrial septum close to the posteromedial commissure of the mitral valve. The tumor had prolapsed into the valve orifice, seriously obstructing blood flow through that structure.

From the General Surgical Services of the Massachusetts General Hospital and Department of Surgery of the Harvard Medical School.

Read at the Thirty-seventh Annual Meeting of The American Association for Thoracic Surgery at Chicago, Ill., May 4-7, 1957.

Under the existing circumstances—particularly since we were aware of the disasters which had invariably occurred[16, 24] when impromptu attempts to remove such tumors had been made—it was thought best to postpone removal until, under hypothermia, the left atrium could be opened and the tumor excised deliberately. The first operation was therefore terminated.

A second operation was carried out on June 15. With the patient's body temperature lowered to 26.4° C. (lower than had actually been planned), the venous inflow to the heart

Fig. 1.—Case 1. Preoperative and 2 month postoperative posteroanterior roentgenograms. The preoperative film shows a slightly enlarged heart with a straight border and congestion of the lung roots. The postoperative film shows a return to normal.

Fig. 2.—Case 1. The arrow indicates the point of attachment of the myxoma to the interatrial septum. The inferior pole of the tumor is grooved where it prolapsed through the mitral valve orifice.

was occluded, the left atrium opened, and the tumor removed (Fig. 2). There was total interruption of the circulation for 5½ minutes. Ventricular fibrillation occurred immediately upon removal of the tumor. Normal rhythm was restored only after 2 hours of "cardiac massage" and elevation of patient's body temperature to 27.5° C. The patient's recovery thereafter was uninterrupted.

The patient's postoperative course was comparatively uneventful. He was discharged to convalescent care on the twenty-fourth day; he returned to work as a steel-construction worker 5 months after operation. Twenty-three months (April, 1957) after his initial operation he was entirely well.

The final pathologic report on the tumor removed was myxoma.

This patient illustrates very well the problems of an intracardiac myxoma. This is the most common primary tumor of the heart, accounting for one third of the reported primary cardiac neoplasms. The majority of myxomas occur in the left atrium where they arise from the rim of the fossa ovalis in the interatrial septum. They are usually pedunculated and may cause ball-valve obstruction of the mitral orifice. The result is a clinical picture indistinguishable in most respects from mitral stenosis.[16, 24, 26, 33]

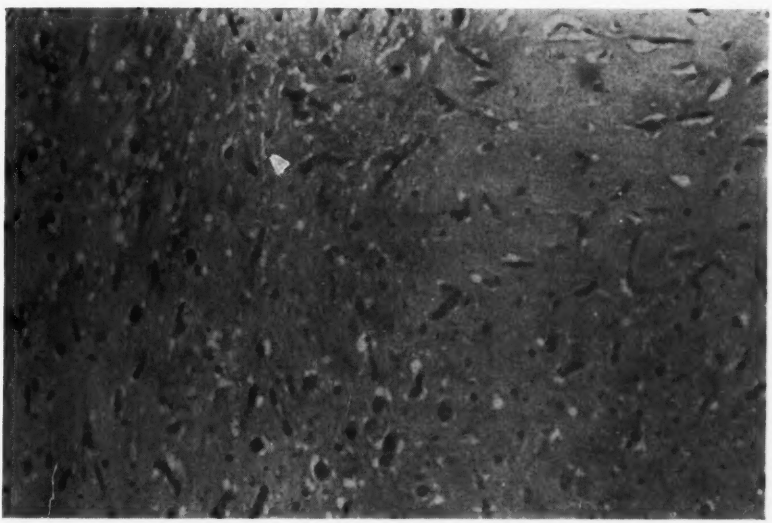

Fig. 3.—Case 1. Myxoma. One of the typical microscopic fields.

Features which suggest intracavitary tumor instead of mitral stenosis are (1) the absence of a history of rheumatic fever, although mitral stenosis without identifiable rheumatic fever in the past is not uncommon, (2) changes in symptoms and murmurs with changes in position, (3) syncope, (4) a rapid progression of symptoms, and (5) marked failure of response to medical therapy.[29, 33] None of these is diagnostic.

If the tumor occurs in the right atrium, obstruction to the tricuspid valve may occur. In this event, one may find the clinical features of tricuspid

disease and signs and symptoms of predominantly right-heart failure. As such, they may closely resemble those of constrictive pericarditis as one of Bahnson's cases demonstrates.[2]

The microscopic picture (Fig. 3) shows a relatively acellular structure of loose connective tissue. The cells vary in size and shape. Some question still remains whether cardiac myxomas are true tumors or organized thrombi. The predominant opinion presently favors neoplasm.[23, 29]

A second case of left atrial myxoma is included here to illustrate several points. In the first place, a change in the character of the murmurs and the occurrence of peripheral arterial emboli in the presence of a normal cardiac rhythm led to a presumptive diagnosis of atrial myxoma. Second, death from cerebral embolus occurred, emphasizing the extreme jeopardy in which such patients live.

Fig. 4.—Case 2. The film shows cardiac enlargement and congestive changes in the lungs.

CASE 2.[11]—The patient was a 9-year-old girl. Eleven months before her final admission, she had had an appendiceal abscess drained. At that time she was found to have an enlarged heart. Two months later, following an interval appendectomy, severe recurrent nosebleeds and low-grade fever led to a provisional diagnosis of rheumatic fever. Salicylates were given and the patient transferred to the House of the Good Samaritan (primarily for children with rheumatic fever).

Three months before admission the patient had a cerebral embolus and an embolus to the index finger of her left hand. There were also several episodes of abdominal pain. Several blood cultures were negative. It was noted that her heart was in regular rhythm. The patient responded poorly to anticoagulant therapy. At this time, atrial tumor was suspected. While arrangements were being completed for angiocardiography in an effort to establish the diagnosis, a second major cerebral embolus occurred from which the patient died (August, 1956).

During the course of the patient's illness the cardiac murmurs changed from a short apical systolic murmur and late diastolic murmur with presystolic accentuation, to a harsh Grade 4 systolic murmur and Grade 3 diastolic rumble. Her chest films (Fig. 4) over a 7-month period of observation showed considerable enlargement of the heart with congestive changes in the lungs.

Autopsy disclosed a gelatinous 5 cm. mass (Fig. 5) attached to but not invading the left atrial wall. The tumor consisted mainly of loose fronds, and tumor emboli were found in the brain, spleen, and kidneys. The final diagnosis was myxoma (Fig. 6).

Fig. 5.—Case 2. The gelatinous myxoma is attached to but not invading the left atrial wall.

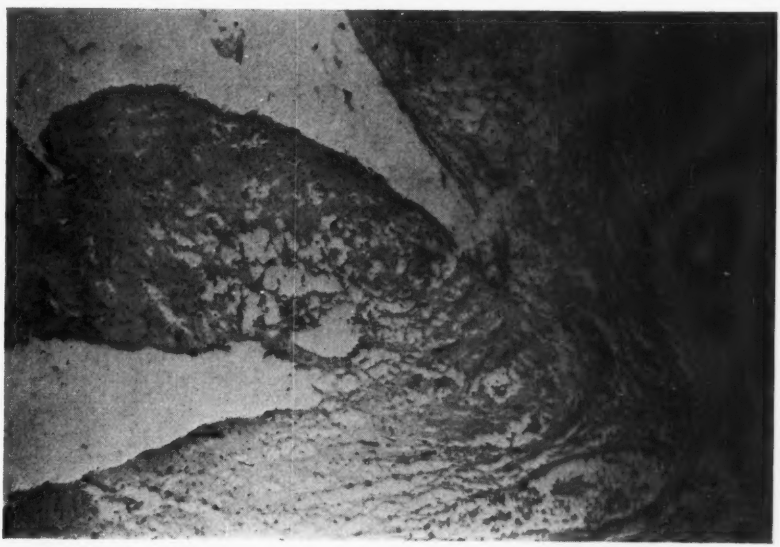

Fig. 6.—Case 2. Myxoma. Microscopic appearance.

The occurrence of tumor emboli from auricular myxomas has been frequently described; in fact, in one reported case, the diagnosis of primary heart tumor was made by pathologic study of the removed peripheral embolus.[11]

Since sudden death from embolization, valvular occlusion, or conduction disturbance is common with cardiac neoplasms, diagnosis and treatment are matters of urgency.

Our third case of primary tumor of the heart is that of a low-grade fibrosarcoma of the right atrium. This tumor was excised successfully and the patient is free of disease one year later.

CASE 3.[10]—The patient was a 7-year-old schoolgirl. Her present illness began 4 months before admission with a severe upper respiratory infection, followed by recurrent bouts of fever. During the week before admission, pain and swelling of the epigastrium, anorexia, fatigue, dyspnea, precordial pain, and puffiness of the face were noted.

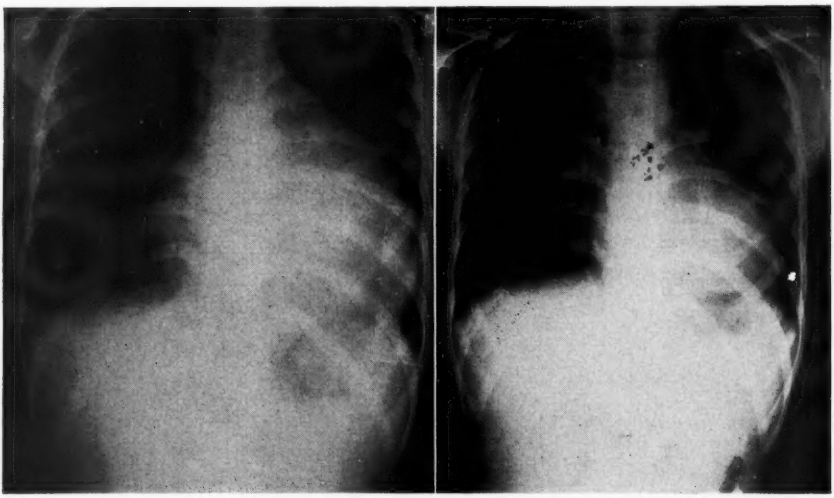

Fig. 7.—Case 3. Posteroanterior roentgenogram before and after pericardiocentesis.

Physical examination, confirmed by x-ray studies (Fig. 7) on admission, was consistent with a sizeable pericardial effusion. On the second hospital day, a pericardiocentesis yielded 400 c.c. of frankly bloody fluid. Following this procedure the patient's clinical state was greatly improved. Chest film (Fig. 7) showed the heart shadow to be almost normal. During the ensuing 2 weeks, x-ray films demonstrated gradual reaccumulation of the pericardial fluid with increasing signs of pericardial tamponade. On the twenty-first hospital day, a second pericardial tap was done with the removal of 400 c.c. of bloody fluid. Cell block and cytologic examination of the fluid were reported negative for tumor, although atypical cells were noted. Clinical improvement again followed pericardial tap, but in the course of the next 10 days, signs of reaccumulation of fluid were apparent and the patient therefore was explored (March 8, 1956) with the purpose of establishing the diagnosis and relieving the tamponade by pericardial-pleural drainage or partial pericardiectomy, if need be.

The pericardial sac was found to be distended by a moderate amount of frankly bloody fluid. As the exploring finger was passed over the anterior surface of the heart, a firm, nodular mass about the size of a walnut was encountered in the region of the right atrial appendage. To visualize this area better, the thoracotomy incision was extended across the sternum. The mass involved the wall of the atrium in the cleft between the appendage and the atrial wall. It was intramural, partly projecting outside the heart, and partly into the right atrial cavity. It was possible to free the tumor sufficiently to apply curved Potts clamps so as to excise the tumor completely with a wedge of atrial wall (Fig. 8). This could be done without interruption of the circulation. The resulting defect in the atrial wall was closed with fine silk, using a flap of atrial appendage to close the gap. The child tolerated the procedure extremely well.

The patient's immediate postoperative course was satisfactory and she was discharged home on the thirteenth hospital day. She re-entered the hospital 2 weeks later with an increasingly rapid pulse and increased venous pressure suggesting recurrent tamponade. The cardiac shadow by x-ray was not particularly enlarged. Under the circumstances, open exploration rather than needle aspiration was considered justified. The operative findings were puzzling in that there was remarkably little pericardial fluid. The heart itself seemed to be somewhat enlarged. There was no evidence of recurrent disease. A large "window" was left in the pericardial sac.

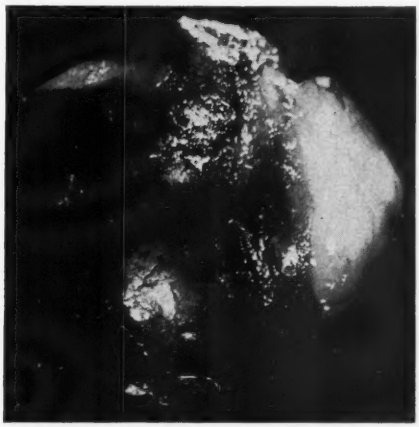

Fig. 8.—Case 3. The dark portion of the sarcoma projected into the pericardial sac, the lighter portion into the atrial cavity. The rim of atrial wall crosses the tumor vertically in this photograph.

The patient's recovery from the second operation was uneventful. One year later (April, 1957), she was reported well without evidence of disease.

A preliminary pathologic report of rhabdomyoma was finally changed to "low-grade fibrosarcoma."

Primary sarcomas of the heart are as variable in histologic types as sarcomas elsewhere. Myxosarcoma, leiomyosarcoma, fibrosarcoma, angiosarcoma, rhabdomyosarcoma and lymphosarcoma have been reported.[23] Taken as a group they are the second most common primary tumors of the heart, forming with myxomas the greatest part of all primary cardiac tumors. Sarcomas may occur anywhere in the heart but are most common on the right side and particularly in the right atrium. Hemopericardium is common. Primary

malignancy as well as secondary malignancy should therefore be considered in the presence of grossly bloody pericardial effusion. Sarcomas are intramural, usually replacing the entire thickness of the heart wall and protruding on one or both surfaces.[29, 32]

This patient did not have any of the commonly described disturbances in heart rhythm nor was the tumor large enough to distort the x-ray outline of the heart.

The tumor was microscopically a low-grade fibrosarcoma (Fig. 9). The cellularity varied in amount and type but most of the cells appeared to be mature fibroblasts. The tumor invaded the atrial muscle in the periphery.

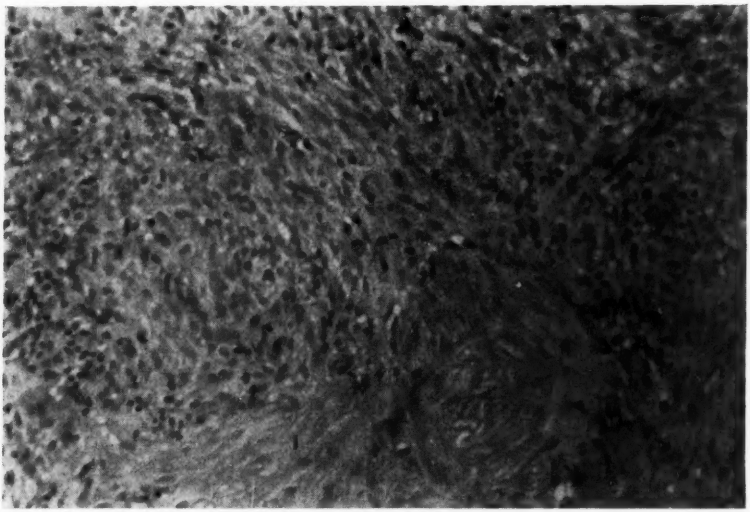

Fig. 9.—Case 3. Fibrosarcoma. Microscopic appearance.

DISCUSSION

Cardiac tumors have been extensively reported in the world literature. The three most useful reviews of the subject are by Yater,[33] in 1931, a comprehensive monograph by Mahaim,[26] in 1945, listing a bibliography of 1,298 references, and an admirable clinicopathologic review by Pritchard,[29] in 1951. Wharton[32] reviewed primary malignant tumors of the heart in 1949. Most of the cardiac tumors reported in the literature are secondary, however. These metastatic lesions characteristically manifest themselves by the appearance of cardiac irregularities in patients with known primary malignancy elsewhere and often widespread metastases.[17] The differential diagnosis from primary cardiac neoplasms is readily made.

As Steinberg and his associates[20, 21, 31] have shown, angiocardiography offers a precise method of visualizing intracardiac tumors on the basis of which careful preoperative planning is possible. Beck[5] demonstrated this 15 years

ago in the case of an intrapericardial, though extracavitary, teratoma, which he resected successfully. At that time he suggested that atrial myxomas might be removed, either by a closed suction method or by open cardiotomy employing a bypass technique.

A number of attempts, many of them successful, to remove intrapericardial tumors and cysts arising from the surface of the heart have been reported. Gebauer,[19] in 1943, attempted to remove an intrapericardial teratoma. D'Abreu,[15] in 1950, removed a hydatid cyst from the wall of the left ventricle. Hochberg and Robinson[22] in the same year excised a hemangioma of the pericardium involving the myocardium. Maurer,[27] in 1952, successfully removed a lobulated intrapericardial lipoma arising from the left ventricular surface. Longino and Meeker,[25] in 1953, partially removed a sarcoma which arose from the right side of the heart and filled the pericardium but the patient died. Bickford, Egan, and Bryce[6] (1956) report the removal of a mesenchymal tumor from the ventricular surface, and Dabbs, Peirce, and Rawson[18] (1957) the successful excision of a large intrapericardial teratoma.

The first successful removal of an intra-atrial myxoma was carried out by Crafoord,[13, 14] in 1954, using extracorporeal circulation and carefully compressing the mitral orifice and the left ventricle while the atrium was widely opened. In 1955, Bigelow[8] encountered a large left atrial tumor during exploration for mitral stenosis. Rather than postpone removal of the tumor to a later operation, as we elected to do, he proceeded to lower the patient's body temperature by flooding the pleural cavity with cool saline. With the cardiac inflow and outflow clamped, it was then possible to remove the tumor through the open atrium. His patient made an excellent recovery. Our patient represents the third successful removal of an intra-atrial myxoma.

Prior to the cases noted above there have been many attempts to remove intracavitary tumors but without final success. Bahnson and Newman[1] nearly succeeded, in 1953, when they removed the major portion of a right atrial myxoma only to have the patient die 3 weeks later. Death was ascribable in part to residual tumor. The other unsuccessful attempts, many of them almost successful, are included among the references cited.[3, 4, 9, 12, 16, 20, 21, 24, 28]

Diagnosis of primary cardiac neoplasms will be made preoperatively in direct proportion to our awareness of their behavior. The knowledge that surgical cure is possible should increase the energy with which these tumors are sought. Once the diagnosis is established, some method of open operation is needed. Almost certainly the use of an extracorporeal pump-oxygenator will be the method of approach in the near future, although hypothermia is not without its value. Blind and hurried attempts to remove such tumors are not to be advocated; although it must be remembered that postponement is not without hazard once the diagnosis has been made.

SUMMARY

Two successful excisions of primary tumors of the heart are presented. One, a myxoma of the left auricle, was removed by cardiotomy under hypothermia. The other, a fibrosarcoma of the right atrium, was removed by partial

auricular excision. A third patient, with a left atrial myxoma, died while awaiting studies. The characteristics and treatment of primary cardiac tumors are discussed.

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Discussion

DR. EGBERT H. FELL, Chicago, Ill.—I wish to congratulate Drs. Scannell and Grillo on this very excellent presentation and on their results. Four years ago we had two attempts at one tumor of the heart with unhappy results that taught us considerable. Thus, the reason for this discussion.

(slide) A 16-year-old boy was sent to us from down state with a diagnosis of possible rheumatic heart disease. Fluoroscopy disclosed a large, partially calcified mass in a very large right atrium which swung back and forth into the entrance of the right ventricle and back to the level of the inferior vena cava. Under hypothermia, we planned to close off the inflow tracts but the inferior vena cava was so short and large that we believed it inadvisable. The examining finger in the right atrium felt a pedunculated tumor about 3 inches long, which bobbed back and forth into the right ventricle and back into the inferior vena cava. It originated on the interatrial wall close to the tricuspid valve. The stalk was smooth and approximately 1 cm. in diameter at its base. The distal end of the tumor was 5 cm. in diameter and of firm consistency. We considered it would be a very easy thing to bring it into a blind pouch. The right ventricular pressure was too high to use a well. A small sterile rubber surgical glove was sutured to the right atrium. As we were opening the atrium the heart stopped and we retreated. Circulation was established by cardiac compression and the blood pressure was maintained for 3 days with Levophed. Six months later we went back in, put in another pouch and the examining finger revealed the tumor to have grown considerably in size; it was very friable and portions separated off the original tumor, causing pulmonary emboli.

(slide) The slide shows the tumor close to the tricuspid valve on the atrial septal side, and it is hanging down into the right ventricle.

(slide) This slide shows some of the friable material broken off.

(slide) This photograph shows the pulmonary artery and lung with tumor in the pulmonary vascular tree, causing the sudden exitus on the table.

In retrospect, we should have made a further attempt to remove the tumor at the first operation. It was not friable and could have been removed then. The second mistake was that we waited 6 months to perform the second operation; however, we did believe there were plenty of reasons for waiting.

DR. WILLIAM A. HOPKINS, Atlanta, Ga.—Dr. Fell has just reported a tumor exactly like the one we want to show. The movement of this tumor was so fantastic by fluoroscopy that we obtained a cinefluorographic picture of this which I shall show you.

This was the case of a 26-year-old man who had symptoms of recurring dyspnea over several years; he had also a persistent tachycardia. As soon as he was examined by x-ray the diagnosis was obvious that he had a calcified tumor within the right auricle. Its location was exactly the same as the one presented before. I should like to show the film.

(film) You can see the movement of this calcified tumor. The lower projection of the tumor as it prolapsed into the right ventricle is not shown as well in this film as in the next section. This patient was prepared and operated on under hypothermia and the tumor was removed successfully. He recovered without complications. As you can see, the movement within the heart is fantastic. This man had a persistent tachycardia, the rate ranging from 110 to 120, and after surgery the rate returned to a normal level. One could feel this tumor project forcefully against the right auricular wall; in diastole it would prolapse into the valvular area of the right ventricle.

DR. ROLLINS HANLON, St. Louis, Mo.—I would like to compliment the essayists on their fine results in these two cases and to present one patient who illustrates several points which I think are relevant.

(slide) Regarding the pathologic aspect of these "tumors," they are, of course, tumors in the sense that they are masses within the heart, but it is my impression that many are not really neoplasms but simply organized clots. This is contrary to the prevailing opinion of pathologists as Dr. Grillo has mentioned. This slide shows a higher magnification of a lesion very similar to the one which he showed as a myxoma. This appears to us to be a thrombus, peculiarly organized because of the forces acting on it in the heart.

(slide) This demonstrates the removal of a mass from the right atrium of a 63-year-old woman with signs of cardiac failure, unaccompanied by cardiac enlargement. Five years after onset of symptoms, the diagnosis was finally established by a variety of studies including angiocardiography. She was intensely cyanotic because the mass blocked the orifice of the tricuspid valve, causing a right-to-left shunt through an atrial septal defect. For several months before operation she had extremely low platelet counts with severe hemarthroses and other hemorrhagic manifestations. The relation of the thrombocytopenia to the "tumor" is of interest; we consider it possible that the intracardiac mass was built up by the agglomeration of platelets and other blood elements.

The mass was removed by open atriotomy with the use of the DeWall pump-oxygenator as shown in this movie, cutting away its attachment to the rim of the inferior vena cava and closing the septal defect with interrupted sutures. The patient has done well in the 6 months since operation with prompt and sustained return of platelets to normal levels.

DR. EDGAR P. MANNIX, JR., Manhasset, N. Y.—This very excellent paper by Drs. Scannell and Grillo emphasizes the importance of establishing an early diagnosis in primary tumors of the heart, inasmuch as they may now be successfully removed surgically. It might be of interest to recall that up to 1955 only 13 in some 350 or more cases of primary tumor of the heart had been diagnosed prior to death. We recently had occasion to see a rather unusual case in a 42-year-old nurse who, for 14 months prior to death, had had a history of dyspnea and progressive heart failure without any previous etiological stigma of heart disease. She subsequently developed cyanosis, clubbing of the fingers and toes and polycythemia, and one month prior to death had a cerebrovascular accident. I saw her for the first time 2 weeks prior to death, at which time she had an enlarged right ventricle by x-ray, right ventricular hypertrophy in the electrocardiogram, and an absent pulmonic second sound. She was in intractable right heart failure. On the basis of these clinical data we assumed that she had either tetralogy of Fallot or pulmonary stenosis with an atrial septal defect. I was unable to obtain an angiocardiogram because of her poor condition. She died without opertion. Autopsy was performed.

(slide) This is the right ventricle greatly hypertrophied and dilated. Obstructing the outflow tract of the right ventricle, in the area of the pulmonary valve, is a huge rhabdomyosarcoma. This lesion was independently diagnosed by three different pathologists as being a rhabdomyosarcoma.

(slide) Here you see a close-up view of this tumor blocking the pulmonary valve, and the little slit on the side is the only point of egress for the blood to get out of the right ventricle into the pulmonary artery.

(slide) This shows a small atrial septal defect, which I think explains why she had cyanosis. We believe that as the pressure rose in the right atrium secondary to right ventricular failure, the patient developed a right-to-left shunt and that the cyanosis was due to this defect plus, of course, the stagnant hypoxia secondary to right heart failure.

Inasmuch as, at autopsy, there was no evidence of metastasis of this lesion we believe that had the diagnosis been established fairly early something constructive might have been done surgically for this patient.

DR. L. FATTI, Johannesburg, South Africa.—Thank you for allowing me to speak before you. I should like to report one tumor from the heart of a patient, without using either hypothermia or an artificial pump.

The patient was a woman aged 45 who showed all the signs of mitral stenosis. Many times differentiation is made between mitral stenosis and myxoma, but I do not think any of the diagnostic points are sufficient to make a firm diagnosis.

At operation the tumor was easily felt and it was evident that the pedicle was a fairly small one. It was a soft gelatinous mass, and the slightest movement threatened to make it move on in the blood stream. It was obvious that the only way to prevent it floating off would be to put a finger into the left ventricle beyond the tumor. A purse-string suture was therefore put into the apex of the heart, a small incision was made and dilated with Hegar's dilators until the index finger of the other hand could be put into the ventricle.

The point here is that the tumor was as large as the mitral orifice, which was twice the size of my index finger. When the pedicle had been cut through with the nail of the atrial finger, it was quite easy to push the tumor from the left ventricle into the left atrium, and as the atrial finger was withdrawn the tumor was pushed into the auricular hole. I had to enlarge the hole in the left atrium a little, give a push and the tumor popped out. In fact it popped out so suddenly that we lost about 20 c.c. of blood. My assistant clamped and closed the hole in the left auricle. I removed the finger from the left ventricle and pulled the purse-string tight.

I think it is a diagnostic point of the nature of the myxoma that it should be so easily cut off from the septum; it is an event that has taken surgeons unaware. But, even if a Brock's knife is used to detach the tumor, the danger of embolus is the same. By this easy method, such disaster is avoided and the operation need not be abandoned. I am told by Logan that he applies a knife to the mitral valve by the same ventricular approach.

DR. ROSS ROBERTSON, Vancouver, B. C.—The paper of Dr. Scannell and Dr. Grillo on primary tumors of the heart interests me intensely. I presented a paper, similarly titled, before the Pacific Coast Surgical Society in February of this year. It will appear in an early issue of the American Journal of Surgery.

Five primary cardiac tumors, treated surgically, were reported. The pathologic diagnoses were teratoma, cylindroma, mesothelial sarcoma, lymphangioma, and myxoma. Two of the patients are apparently cured, one was improved for several years, and 2 died postoperatively. The 2 survivors apply directly to this discussion.

(slide) The first was a 10-year-old boy who complained of dyspnea on exertion. Cardiac x-rays showed a rounded area of density with calcification in the anterior mediastinum, deeply indenting the right atrium and displacing the pulmonary artery and aorta, as in the cases described by Dr. Dabbs yesterday.

(slide) Removal of the tumor was difficult because of its close attachment to the aorta. It received a small branch from the right coronary artery. Pathologic diagnosis: Cystic lymphangioma.

(slide) This patient was explored for mitral stenosis. A myxoma was found in the left auricle. The mitral valve was normal. The wound was closed and a second operation was planned. In retrospect, there were some elements in the history, as described by Dr. Grillo, which pointed to the correct diagnosis.

(slide) At the second operation, under hypothermia, the venae cavae were taped, the descending aorta was clamped and the coronary arteries were perfused with oxygenated blood through a cannula in the internal mammary artery, a method similar to that of Dr. Maloney. The tumor, the size of a golf ball, was removed in two pieces. It was attached to the auricular septum. No residual tumor could be seen.

(slide) The patient has made an excellent recovery, as shown in this postoperative chest film.

(slide) Routine sections showed myxomatous tissue containing many hemorrhagic areas.

(slide) Because the hemorrhagic areas simulated to some extent the early organization of the thrombus, our pathologist examined a number of thrombi, both human and experimental, for comparison. In sections stained with colloidal iron, the tumor was strongly positive for acid mucopolysaccharides, as shown here. The controls were negative.

DR. J. GORDON SCANNELL, Boston, Mass. (Closing).—I should like to thank very much those who have discussed the paper. I must say that the cineradiographic pictures of an intracardiac tumor in action are most dramatic and not a little alarming. The pathologic considerations that Dr. Hanlon has brought up are very interesting. We have discussed the matter at length with the members of our department of pathology and have accepted their considered opinion that these probably represent tumors although certainly the idea that they may represent organized thrombi is well rooted in the literature. As you know, many of these were originally called polyps of the heart.

In closing, I would like to emphasize one point in particular. In most cases of myxoma of the heart, the diagnosis is going to be made in the course of an operation for mitral stenosis. Ideally, the surgeon should proceed at this time with open operation and removal of the tumor. If he is not in a position to do this, whether by cardiac bypass or hypothermia, then it is our conviction that it would be better to postpone final attack upon the tumor to another day. Admittedly, this is a calculated risk, but at the present stage of affairs, we believe it is a justified one.

VENTRICULOPLASTY FOR CARDIAC ANEURYSM

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CARDIAC aneurysm usually involves the left ventricle and commonly is associated with certain physiologic and clinical features which bode ill with respect to the patient's welfare and longevity. Etiologically ventricular aneurysms are of three types: (1) those due to congenital malformation—often described under the term "diverticulum," (2) those resulting from trauma (nowadays usually produced by cardiac surgery), in which case the sac usually is formed by certain extracardiac structures, especially the pericardium (false aneurysm), and (3) those resulting from weakening and bulging of a relatively localized area of myomalacia of the heart wall. These latter most often result from acute myocardial infarction. However, localized forms of syphilis,[1, 2] tuberculous caseation,[3] nonspecific mediastinal abscess,[4] and even the anomalous origin of a coronary artery from the pulmonary artery[5] have been indicted as causative of ventricular aneurysm.

The actual incidence of aneurysm formation following myocardial infarction is subject to considerable dispute, various authors placing it as low as 5 per cent,[6] while others consider the rate of development of this complication to be of the order of 20 per cent.[5, 7] The world literature generally establishes it at about 10 per cent.[8, 9, 10] Part of this controversy devolves about the criteria of definition, some[5, 11] considering that any enlargement of the ventricular lumen due to a localized encroachment upon the thickness of the ventricular wall constitutes an aneurysm. Others restrict the term to those grosser deformations of the heart which produce an obvious outward bulging of the ventricular outline (Fig. 1). Naturally these latter lesions are characterized by a more severe physiologic abnormality and by a more rapidly progressive downward clinical course.

Whether the incidence finally be established at 5 or 20 per cent, the high current rate of occurrence of acute myocardial infarction which is estimated variously by Master[12] at 500,000 to 800,000 per year in the United States suggests that between 25,000 and 160,000 new cases of ventricular aneurysm develop annually in this one country. Since the study of Schlicter, Hellerstein, and Katz[5] places the average life span (with invalidism) following the appearance of aneurysm at somewhat over 2 years, there should be at least twice as many of these patients alive at any given moment. The miserable and progressively downhill course which most of them manifest with conservative

This work was supported by the Mary Bailey Foundation for Heart and Great Vessel Research, Philadelphia, Pa.

Read at the Thirty-seventh Annual Meeting of The American Association for Thoracic Surgery at Chicago, Ill., May 4-7, 1957.

management suggests that every effort should be made to improve the medical and surgical armamentarium available for the treatment of this disease. It is the purpose of the authors to show that an effective and generally satisfactory method of surgical attack for this condition now exists. To date, 9 patients with ventricular aneurysm have been operated by us, and 8 have survived in a much improved condition.

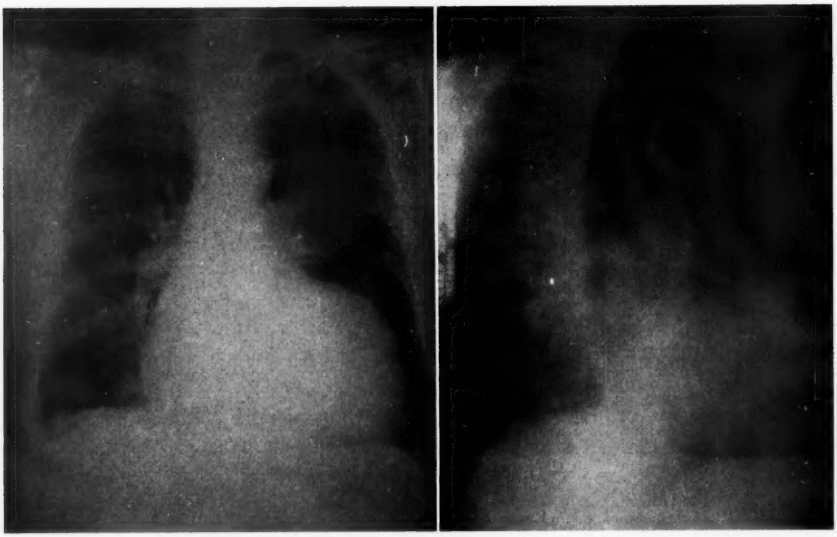


Fig. 1.—PA and oblique roentgenograms obtained from J. L., a patient with huge indisputable left ventricular (postinfarctional) aneurysm.

CONGENITAL DIVERTICULUM OF THE VENTRICLE

This condition would seem to qualify by all logical criteria as a ventricular aneurysm. It exists at birth as a muscular outpouching usually of the left, but occasionally of the right ventricle, or even of both. Since there usually is free luminal communication with the indicated ventricle the latter type of lesion (open to both ventricles) may permit a physiologic shunt of blood at this level. Other than this relatively rare communicating lesion there characteristically is no physiologic abnormality associated with a congenital ventricular diverticulum since the muscular walls are continuous with and contract synchronously with the ventricles.

The chief danger in these cases is that the abnormal muscular outpouching eventually undergoes rupture with resulting fatality. The total (although limited) world experience with this uncommon lesion indicates that it is best treated by surgical excision at an early age.[13-20] An illustrative report of a personal case follows.

CASE 1.—K. W. (Hospital No. 54-01397), a 7-day-old white female infant, was admitted to the pediatric service of the Hahnemann Hospital in Philadelphia on Feb. 4, 1954. Her

complaints were those of a feeding difficulty but it had been noticed that there was an abnormal, obliquely directed mass in the epigastric region which pulsated synchronously with the cardiac contractions (Fig. 2). The overlying skin was thin and abnormal, suggesting a central defect of union of the bodily segments. Angiocardiography showed a communication between the left ventricle and an abnormal blood-containing chamber. The diagnosis of congenital diverticulum of the ventricle was made and operation was carried out on Feb. 11, 1954. The mass was dissected free of the overlying abnormal skin and adjacent fibromuscular tissues. It was found to be covered with an extension of the pericardial sac and to be directly continuous with the apex of the heart. There was no opening into either the peritoneal or pleural cavities. The apparent site of junction of the mass with the ventricle was cross-clamped with a curved pair of Potts multiple-toothed forceps. The mass continued visibly to contract in synchronism with the ventricles.

The diverticulum was amputated along a line 5 mm. distal to the clamp (Fig. 3) and the cut end was oversewn using one row of continuous mattress sutures of 3-0 cotton with a swedged-on curved needle. Free luminal communication was demonstrable. A second running suture was used to complete the repair. The clamp was removed and the abdominal wall was closed anatomically.

The postoperative course was uneventful, and the infant was discharged from the hospital in good condition.

TRAUMATIC ANEURYSM OF THE VENTRICLE

Accidental injuries, particularly those of a gunshot or stab wound type, may be counted upon to create an occasional aneurysm of the ventricular wall. However, the rapidly increasing incidence of surgical ventriculotomies (with a certain percentage of poor healing) promises a considerably larger harvest of ventricular aneurysms of traumatic origin in the near future. Probably only the fact that most ventriculotomies are made into the relatively low tensioned right ventricle prevents the rate of aneurysm development from being strikingly high. Brock[21] has mentioned this as a complication of pulmonary valvulotomy and infundibulectomy. Indubitably, meticulous care in the performance of the ventricular repair will tend to reduce the incidence of the complication. This is especially pertinent with respect to the larger ventriculotomies such as are employed in current "open-heart" techniques.

Undoubtedly, the first step in the mechanism of development of such an aneurysm is a partial dehiscence of the deeper layers of the myocardial line of closure. In many instances, this may be due to failure to appose accurately the inner aspect of the lips of the incision, perhaps due to failure to apply the sutures deeply enough. Thus, a site for beginning dissection of the wound surfaces is provided. This mechanism would seem to be the determinative cause in both cases of traumatic aneurysm which are cited in this presentation. In each, a relatively small left ventricular wound was closed rapidly with surface approximating sutures, the smallness of the ventricular opening preventing certainty of inclusion of endocardial tissue within each of the individual sutures.

Other factors, more difficult to assay, include metabolic deficiencies, hypertension within the ventricular chamber, hypoproteinemia, anemia, and certain specific and nonspecific degenerative diseases of the myocardium.

It would seem probable that a beginning dissection of the cardiac wound would require at least several days for completion, thus enabling reinforcing

Fig. 2.—Photograph of infant, K. W. (Case 1), showing obliquely directed pulsating epigastric mass and abnormal line of skin fusion. (Bailey, Surgery of the Heart, ed. 1, Philadelphia, 1955, Lea & Febiger, p. 408.)

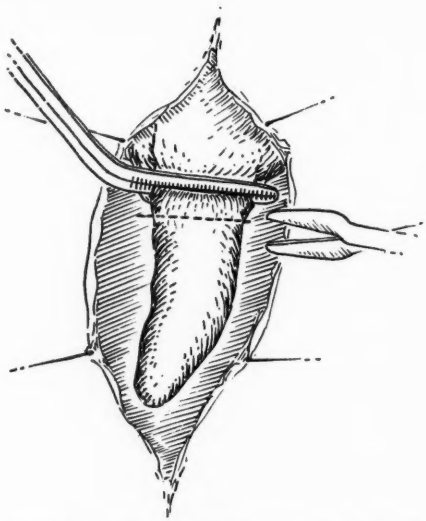


Fig. 3.—Line of indicated section (dotted). (Bailey, Surgery of the Heart, ed. 1, Philadelphia, 1955, Lea & Febiger, p. 411.)

adhesions to develop between the surface of the heart and the overlying pericardium (when it has been reconstructed) or other contiguous justacardiac surface (mediastinal tissues, sternum, lung, etc.). Further dissection and stretching of these apposed adherent tissues will serve to create a sizable aneurysmal sac of false type. In most instances, this will communicate with the left ventricular lumen by a relatively narrow transmural channel. Sometimes the communication may be multiple, as in the first of our personal cases (Case 2). Whether any or much thrombosis will develop within such a false aneurysmal sac depends upon the size of the junctional passage and upon the freedom of blood ingress and egress during each cardiac cycle. In Case 2, as soon as the exploring finger tip was applied in such a way as to block blood flow through the larger of the two luminal communications, thus greatly reducing the amount of ebb-and-flow, the liquid blood content of the sac promptly became coagulated (within 4 minutes).

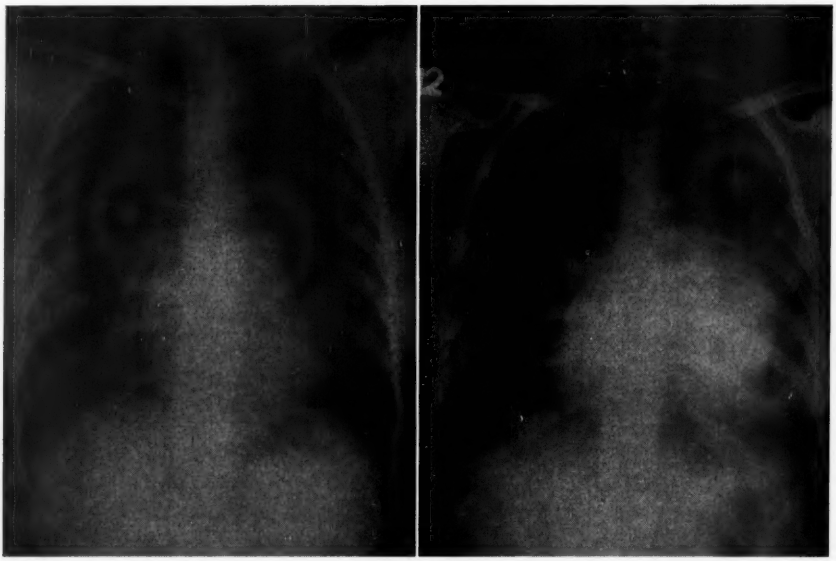
Such false aneurysms of traumatic origin may be presumed to superimpose some additional physiologic burden upon the left ventricle, since the semielastic aneurysm visibly becomes distended during systole and tends to collapse partially during diastole. In other words, a certain amount of paradoxical function is imposed upon the left ventricle thus reducing the efficiency of its gross stroke output. However, because of the usual small size of the communicating transmural passage the effective loss of output characteristically is not great and can be borne for a considerable period of time. However, one of our 2 patients (E. S., Case 3) exhibited signs and symptoms of serious lowering of myocardial reserve at the time of secondary surgery.

Should massive or obliterative thrombosis reduce the aneurysmal lumen the effect would be to diminish the amount of the paradox and thus to lessen the physiologic burden. Complete cure by such a mechanism is not inconceivable although presumably it is improbable. The likelihood of arterial embolization, due to extension of the thrombotic process into the ventricular lumen or to detachment of a loose fragment from the sac, would seem to be small in these cases again because of the usual narrowness of the transmural channel.

Indeed the greatest hazards to the patient with traumatic (false) aneurysm of the ventricle would seem to be the twin probabilities of continued growth of the sac (causing pressure symptoms) and its tendency to eventual rupture. The likelihood and imminence of such developments would seem to be dependent upon the tightness of the restricting adhesions, the tensile strength of the tissues making up the false sac, the size of the ventricular communication, and the level of intraventricular pressure during systole.[22]

CASE 2.—G. F. (Hospital No. 55-10624), a 31-year-old white housewife was admitted to the Hahnemann Hospital in Philadelphia on Sept. 16, 1955, for investigation of an abnormal shadow evident upon the left cardiac border. This had developed shortly after a mitral commissurotomy had been attempted at another clinic 4 months previously. On that occasion (May 8, 1955), the wall of the left ventricle inadvertently had been perforated from within by the index finger of the operating surgeon. This had resulted in massive blood loss and the onset of ventricular fibrillation. The attempt at commissurotomy was terminated promptly, hasty suture repair of the laceration was carried out, and the heart was defibrillated successfully with electric countershock.

The postoperative course was stormy but finally the patient was discharged in good condition with clinical relief of the symptoms caused by the mitral obstruction. However, in August, 1955, an abnormal bulging shadow was observed along the left cardiac border (Fig. 4, *A* and *B*). In view of the history it was suspected that this might be a ventricular aneurysm. During the next few weeks the shadow was seen radiographically to increase in size, and the patient began to complain of precordial discomfort.



A. *B.*

Fig. 4.—*A*, PA roentgenogram of G. F. (Case 2) taken prior to first commissurotomy attempt.

B, PA roentgenogram of G. F. taken a few weeks after inadvertent laceration of the left ventricle. The abnormal shadow was suspected of being a ventricular aneurysm. (Smith and associates, Surgery 42: 497, 1957.)

The patient had a normal sinus rhythm, a sharp first sound at the apex, and a mid-late rumbling diastolic murmur. The pulmonary second sound was accentuated.

The electrocardiogram showed an elevation of the S-T segment in Leads I, AVR, and AVL. There was S-T segment depression in Leads II, III, AVF, and V_4 through V_6. The T wave was inverted in Leads I and AVL. The tracings were considered suggestive of damage to the high lateral wall of the left ventricle.

The x-ray investigation included kymographic studies which indicated pulsations in the mass of 10 mm. amplitude and these were paradoxical to ventricular systole. Cardioangiography[23-28] was carried out by direct puncture of the left atrium, demonstrating a narrow communication between the left ventricle and an opacified cavity within the "bulge." The diagnosis of ventricular aneurysm of false type was deemed confirmed.

At operation on Sept. 27, 1955, the mass was seen to be contained within and to be formed by a greatly thickened and adherent pericardium. A purse-string suture was placed about a selected site over the fundus of the sac, and the operator's ungloved left index finger was inserted through a stab wound made within its confines. The sac was found to be free of clot and to communicate with the left ventricular lumen by two apertures separated by a few millimeters of ventricular wall from each of which a jet of blood emerged with ventricular systole.

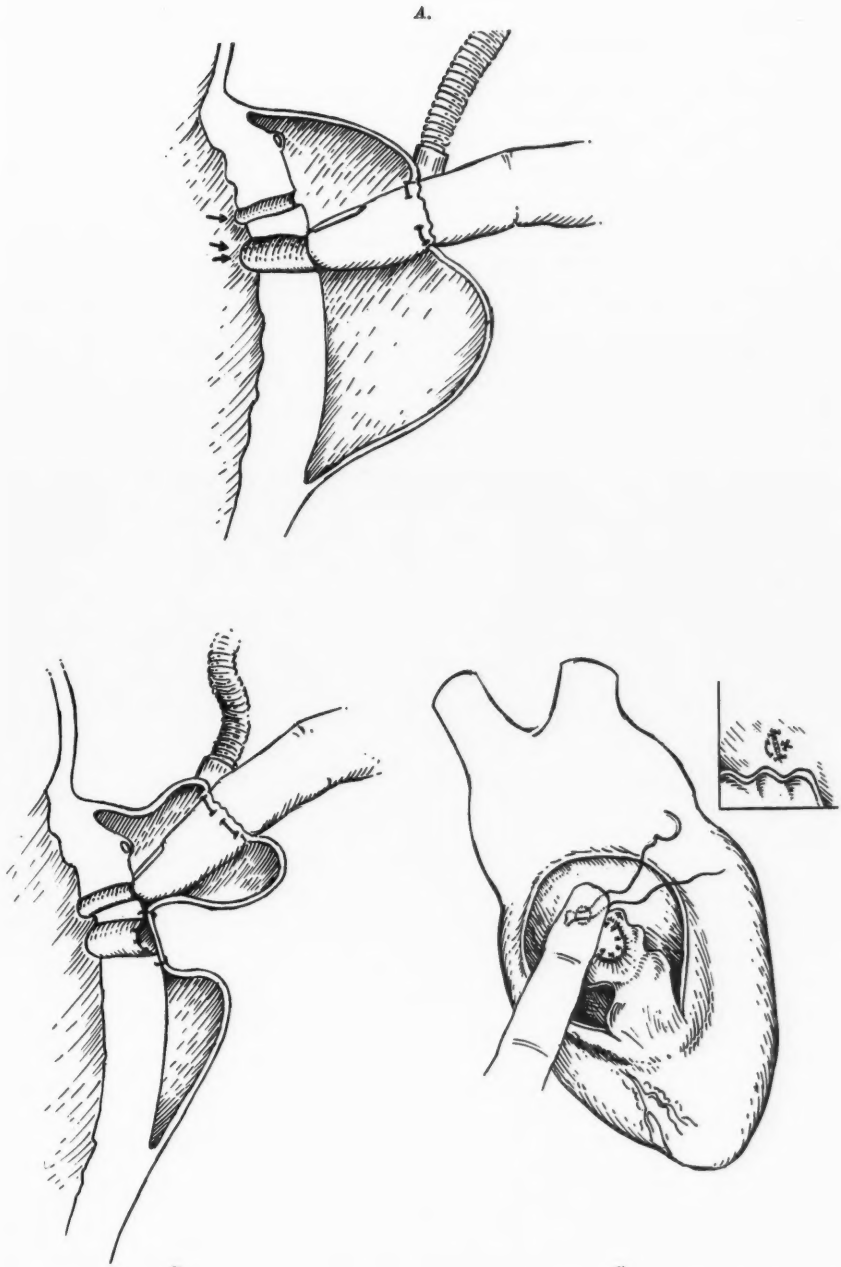


Fig. 5.—*A*, Diagrammatic representation of method digitally examining interior of pseudoaneurysm in G. F. There were two openings into the left ventricle from each of which a jet of blood could be felt to emerge with each ventricular systole.

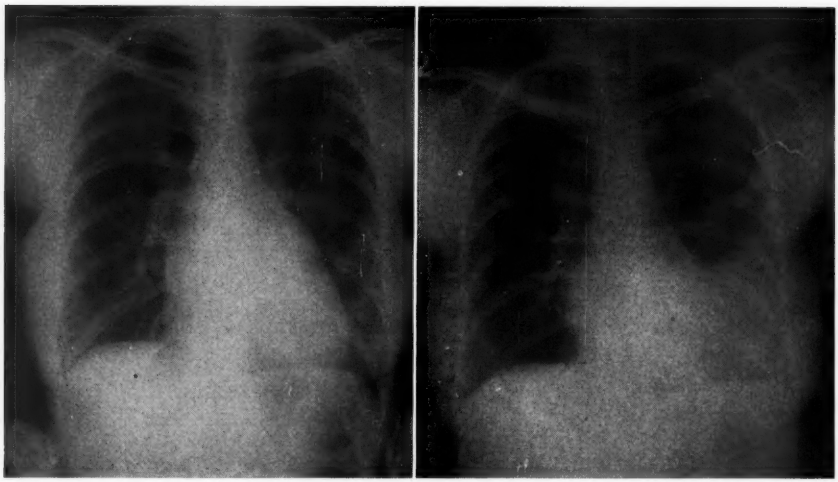
B, The flexible aneurysmal wall was sutured down upon the lower opening in order to block it temporarily.

C, Using the tip of the intra-aneurysmal finger, the upper opening was tamponaded while the sac was opened widely. Then this transventricular passage was closed definitively by passing suturing needles under the finger tip, as shown.

(Smith and associates, Surgery 42: 500-501, 1957.)

The exploring finger tip was applied over the lower (larger) opening, blocking its jet. Within a few moments the blood within the sac became coagulated. Temporary sutures of silk were applied between the wall of the sac and the myocardial surface surrounding the lower opening. When they were tied down, the opening became blocked in exactly the same way that an atrial septal defect is closed by suture approximation of its margins to the overlying atrial wall (atrioseptopexy).[29-33]

Now the upper opening was tamponaded with the finger tip and the remaining lumen of the sac was opened boldly. Deep approximating mattress sutures, swedged on curved needles, were applied beneath the finger tip to close this ventricular aperture definitively. The temporary sutures blocking the lower opening were cut away, and this remaining passage was tamponaded digitally. It was sutured then in the same manner as the first (Fig. 5, *A*, *B*, *C*). After repairing the ventricular wall, the mitral valve was explored through an opening made in the appendageal stump and an area of fusion found in the region of the anterior commissure was sectioned with the guillotine knife. Recovery was uneventful and the patient has remained well to date.



A. *B.*

Fig. 6.—*A*, PA roentgenogram taken on E. S. (Case 3) prior to surgery.
B, Marked abnormality of cardiac outline in E. S. observed one year after aortic surgery. (Smith and associates, Surgery, 42: 503, 1957.)

CASE 3.—E. S. (Hospital No. 55-14112), a 38-year-old white woman, was operated upon at the Hahnemann Hospital in Philadelphia on Nov. 30, 1953, for combined mitral and aortic stenosis, having the then available transventricular instrumental approach used for the latter valve. Recovery seemed satisfactory and the patient remained in apparent good health until she was involved in an auto accident in July, 1955.

Shortly thereafter she developed atrial fibrillation. X-ray studies at that time showed an abnormal bulge over the left border of the cardiac silhouette. In retrospect, it was decided that a similar but smaller shadow had existed in an early postoperative film (Fig. 6, *A* and *B*).

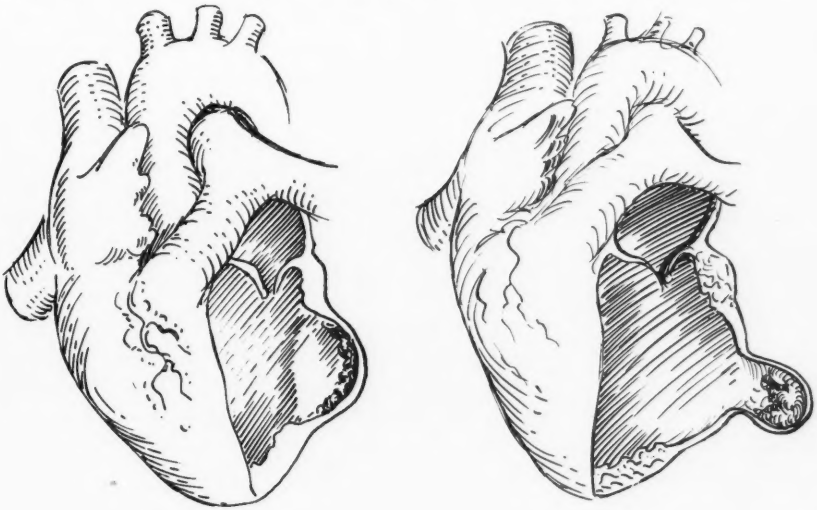
During the next 3 months, the bulge increased in size and the patient began to suffer from frequent attacks of paroxysmal nocturnal dyspnea. She was re-admitted to the Hahnemann Hospital on Dec. 6, 1955, with a diagnosis of left ventricular aneurysm.

Clinical and physiologic studies revealed a harsh systolic murmur and an early blowing diastolic murmur in the aortic area, and a sharp first sound at the apex (but no mid-late diastolic murmur). The electrocardiogram showed T-wave inversion in Leads I and AVL

suggestive of myocardial damage high on the lateral wall of the left ventricle. The left ventricular pressure was 140/10 mm. Hg, the left atrial (mean) pressure was 25 mm. Hg, and the aortic pressure was 126/84. It was felt that mitral stenosis existed, but that no serious obstruction existed at the aortic level.

Cardioangiography by direct left atrial puncture showed an abnormal cavity communicating with the left ventricular chamber.

At operation on Dec. 16, 1955, an ovoid expansile (pulsating) mass the size of a lemon was seen to arise from the left ventricle in the region of the previous operative intervention. It lay beneath and was largely composed of thickened pericardium. The operator's ungloved left index finger was inserted into its lumen through a purse-stringed stab wound as in Case 2. A single communication into the ventricle was recognized. It was tamponaded with the



A. *B.*

Fig. 7.—*A*, Diagrammatic representation of diffuse aneurysmal bulge which is the type which characteristically develops in the acute stages of a myocardial infarction. Intraluminal clotting is common.

B, Representation of the thin-walled narrow-necked aneurysm which is characteristic of those which develop late in the course of healing of an infarcted area or as the result of a secondary infarction. Thrombosis is usual within this type of sac.

finger tip, the sac was cut away, and the ventricular wall defect was closed with mattress sutures. The mitral valve was re-explored through an incision made in the stump of the left auricular appendage. The valve would admit but a finger and therefore was sectioned at both commissures. Recovery was uneventful.

POSTINFARCTIONAL ANEURYSMS

This group comprises by far the commonest and most important of these lesions. To date, we have operated upon 6 such patients with 5 long-term survivals, including the first historically to recover from excision of a ventricular aneurysm. This case has been reported elsewhere[34] and the operative technique has been described under the very appropriate term of "ventriculoplasty."[34] The abnormality in these cases varies from a relatively diffuse bulging of a portion of the ventricular wall to a thin-walled, pouchlike or balloonlike structure which communicates with the ventricle by a narrow neck (Fig. 7, *A* and *B*). The former type is the one which develops in the acute

stages of a myocardial infarction, and is characterized by the presence of muscular elements in its relatively thick wall. The latter type is a product of the later or chronic stages of a myocardial infarction and is composed chiefly of scar tissue. In either case, the sac may contain clotted material which sometimes becomes calcified. Schlicter, Hellerstein, and Katz[5] found thrombosis in over 50 per cent of their series. This was associated with a 35 per cent incidence of thromboembolic episodes.

The commonest site of localization is the anterolateral aspect of the left ventricle with involvement of the apex in over 50 per cent of the cases. The next most common is the posterodiaphragmatic surface (about 25 per cent). Aneurysmal bulge of the septum is less frequent. The right ventricle is least often involved. Effective treatment of the septal type of lesion would seem to require an open technique with a cardiopulmonary bypass for a proper repair. In this connection, it may be noted that both Cooley[35] and Effler[36] have sutured an acquired defect of the interventricular septum caused by rupture following infarction.

The localization and configurations in our 6 cases were as follows: 2 anterolateral with diffuse bulging including the apex, one localized lateral near the atrioventricular groove, one posterodiaphragmatic, and one huge diffuse lateral mass. There was one double sac with anterolateral and apical bulges.

PATHOPHYSIOLOGY

Due to the semielastic structure of the sac, it becomes stretched out at the height of systole (paradoxical pulsation). The ventricular contraction must raise the intraluminal tension of the sac above the level of the aortic diastolic pressure before ejection can take place. Thus, the dysfunction partakes of that found in mitral regurgitation, a portion of the ventricular content being "wasted" during each beat in distending the abnormal chamber rather than being expelled into the aorta. During the diastolic period, the semielastic sac empties much of its content into the left ventricle, impeding filling from the left atrium. In this respect, the abnormal physiology resembles that of aortic regurgitation.

As a result of this functional inefficiency, the left ventricle is overworked and gradually undergoes compensatory dilatation and hypertrophy. This very enlargement, as Burch and co-workers[37] have shown mathematically, greatly enhances the mechanical handicap. Furthermore, it would seem that the ballooning and outpouching of the aneurysm may exercise a stretching effect upon the adjacent portions of the myocardium during systole, thus contributing to an abnormal and relatively ineffective type of ventricular contraction.

PROGNOSIS

While some aneurysms of the ventricle may be silent and, for a time, pass unnoticed clinically, this is true in less than half the cases. In them, the unexpected onset of arterial embolism may be the first indication of the presence of the lesion. In most cases, the pain of coronary insufficiency persists after the infarction, heart failure develops, and embolic issue or sudden death may punctuate the picture. Schlicter and associates[5] in their study of 102 patients reported a 70 per cent incidence of heart failure, this being the main cause of

death in 48.1 per cent. Embolism occurred in 35 per cent and was the main cause of death in 21.7 per cent. Three fourths of their patients died within 3 years, and 88 per cent within 5 years. Actual rupture of the aneurysm is rare,[5, 10, 38, 39, 40] probably because death occurs from some other cause before the dilatation and thinning of the sac becomes extreme. This outlook is dismal in contrast to the average life span of more than 12 years and to the 90 per cent of complete rehabilitation to be expected after uncomplicated myocardial infarction.[39, 40, 41, 45]

DIAGNOSIS

While certain physical signs and certain electrocardiographic findings[5, 39, 41] are considered suggestive of the presence of a ventricular aneurysm, it is our belief that the best and most reliable diagnostic means are radiologic. These include the classic fluoroscopic findings (paradoxical pulsating mass on the border of the cardiac shadow), simple radiographic disclosure of a suggestive

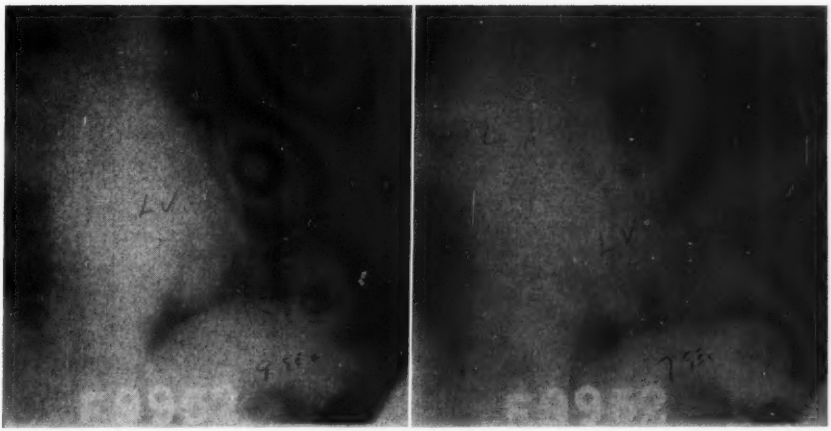
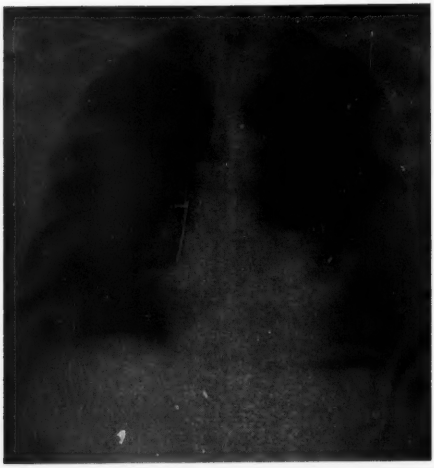


Fig. 8.—By opacification techniques it often is possible to demonstrate the presence and amount of thrombosis which exists within a ventricular aneurysm. (Same case as Fig. 1.)

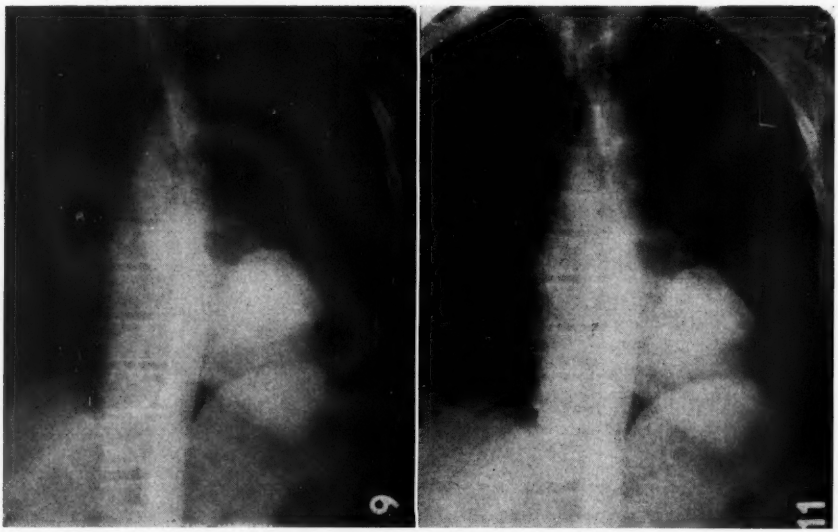
bulge upon the cardiac contour, and roentgenkymography. However, it appears that radiographic identification of the opacified left ventricle, preferably by direct left intra-atrial or left intraventricular injection (ventriculography)[23-28] is the surest method of establishing the diagnosis (Figs. 8 and 9). By these techniques, not only the exact anatomic localization of the mass but also the extent and type of involvement and the presence or absence of massive intraluminal thrombosis may be established. The diagnosis of ventricular aneurysm was confirmed in this manner in all but Case 4, in which it seemed evident enough by ordinary radiographic examination.

SURGICAL TREATMENT

In each of our cases, this amounted simply to a subtotal resection of the aneurysm without the use of a cardiopulmonary bypass, enough of the mixed muscular and fibrosed basal tissue of the scar of healing being preserved to



A.



B.

Fig. 9.—Demonstration of a postinfarctional aneurysm later corrected by ventriculoplasty in S. C., a 41-year-old white man. *A,* PA roentgenogram showing nondescript shadow along left ventricular border.

B, Opacification of left ventricle by direct ventricular injection shows an unusual communicating shadow with a flattened top.

permit reconstruction of a ventricular chamber of approximately the normal size and shape for the particular individual. Hence, the term "ventriculoplasty." However, these simple statements compress a great many technical details and the results of considerable clinical, physiologic, and experimental investigation which have not yet been disseminated widely to the profession. That this information may be surgically important is suggested by the fact that we are aware of only 2 long-term survivals among a number of resections for ventricular aneurysm which have been performed by other thoracic surgeons throughout the world. Indeed, some surgeons have chosen to carry out some type of enfolding or plicating operation upon the wall of the sac[43-46] rather than to undertake the presumed great risk of resection.[47-51]

By word of mouth, we have been informed that the causes of death in these clinical cases largely have been of two types: (1) Arterial embolization occurring at the time of operation. Our own single mortality was caused by gross embolization at the moment of compression of the base of the aneurysmal sac in a patient who was known (see Fig. 8) to have had massive clotting within the sac. Since that time, we have worked out a simple and practicable method for the avoidance of this complication. (2) Congestive heart failure during the weeks or months immediately following the operative intervention. While many of these patients may have been in a state of incompletely relieved congestive failure at the time of operation, our experience to date has indicated[50, 51] that immediate improvement is to be expected in this respect following removal of the severe physiologic handicap imposed by the functional existence of the paradoxically expansile mass. True, in some, progression or a fortuitous exacerbation of the underlying arterial or myocardial disease might have contributed to a continuation of the downward course. However, it is our belief that in many cases the heart failure must be attributed to a technical error on the surgeon's part. It may be that inadvertently he has included one or more important coronary arterial branches within the suture line, thus aggravating the existing myocardial ischemia. However, it is more probable that his surgical sin has been that of excessive removal of ventricular wall tissue with consequent severe reduction in the size of the left ventricular cavity!

This may seem to be a rather surprising statement since most physicians feel that the cardiac chambers are capable of prompt and extensive compensatory dilatation. While this undoubtedly is true under certain clinical conditions, it appears definitely to be untrue with respect to surgical resecion of the ventricular wall. This was brought home to us by the results of an experimental study of the treatment of acute myocardial infarction by excision as suggested by Murray.[52]

EXPERIMENTAL METHOD

Tennant and Wiggers[53] noted that the acutely infarcted area of the heart would beat paradoxically. Murray,[52] in his now classical article, demonstrated that the area could be resected with survival. This acute method of producing a paradoxically beating area in the heart led us to reproduce these experiments in an attempt to solve the problems attendant upon the resection of ventricular

aneurysms. They also led to an interesting observation on the animals when large areas of acutely infarcted muscle were removed, which was in contrast to the previous work.

Fig. 10.—Photograph showing the cyanotic infarcted paradoxically pulsating area of left ventricular wall which develops following ligation of the anterior descending branch of the left coronary artery in the dog.

Fig. 11.—Application of the Rumel-Morse clamps armed with strips of Ivalon sponge to exclude the infarcted portion of the left ventricle.

Initial attempts were made simply to place mattress sutures under the infarcted area, tie them down, and then excise the infarcted area. It was noted that when a large area such as that produced by ligation of the anterior descending branch of the left coronary artery was involved, the tension necessary

to coapt the edges would cause them to cut through the muscle with consequent severe hemorrhage. Further, there was no provision in this technique for the evacuation of clots such as might occur in aneurysms. Therefore, a method for evacuation of clots and for buffering the suture line was devised.

Twenty-five mongrel dogs, weighing from 5 to 15 Kg., were prepared. The anterior descending coronary artery was ligated 2 to 3 cm. from the point where it branched from the main left coronary. This site was chosen to avoid possible inclusion of the septal artery. The subsequent infarction involved at least one half of the anterior surface of the left ventricle and the apex (Fig. 10). A Rumel ventricular clamp, as modified by Morse,[54] was armed with Ivalon strips placed so that the teeth protruded through the sponge (Fig. 11).

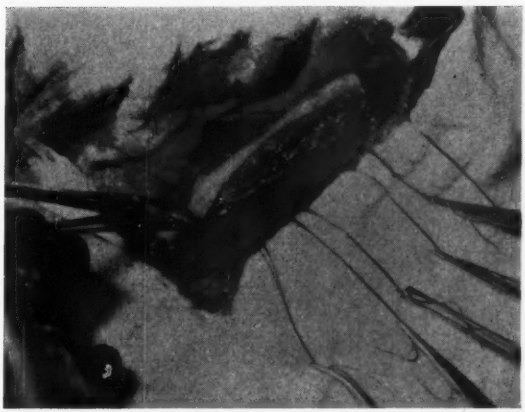


Fig. 12.—The approximating sutures are passed through the Ivalon strips after amputating the infarcted muscle.

These were applied at the limits of the infarcted area into the normal muscle so that the teeth were engaged into but did not penetrate the wall. A large incision was made into the infarcted area and the wound was allowed momentarily to bleed. By this method any contained clots would have been "flushed-out." The bleeding could be controlled instantaneously and completely by closing the clamp. The Ivalon sponge prevented its jaws from cutting through the wall or injuring it. The clamp was undersewn, using interrupted, overlapping mattress sutures placed directly through the Ivalon. The excluded portion of the ventricle then was cut off above the sutures. The clamp was removed and the Ivalon sponge left permanently in place (Fig. 12). If the mattress sutures are placed properly so that they overlap, no additional sutures are needed. This method appears to overcome most of the technical problems concerned with the resection of a ventricular aneurysm.

As the idea of excision of areas of myocardial infarction has been postulated and the opinion exists that the time for resection may not be after the aneurysm has formed but immediately after the acute infarction, the dogs were saved for further study.

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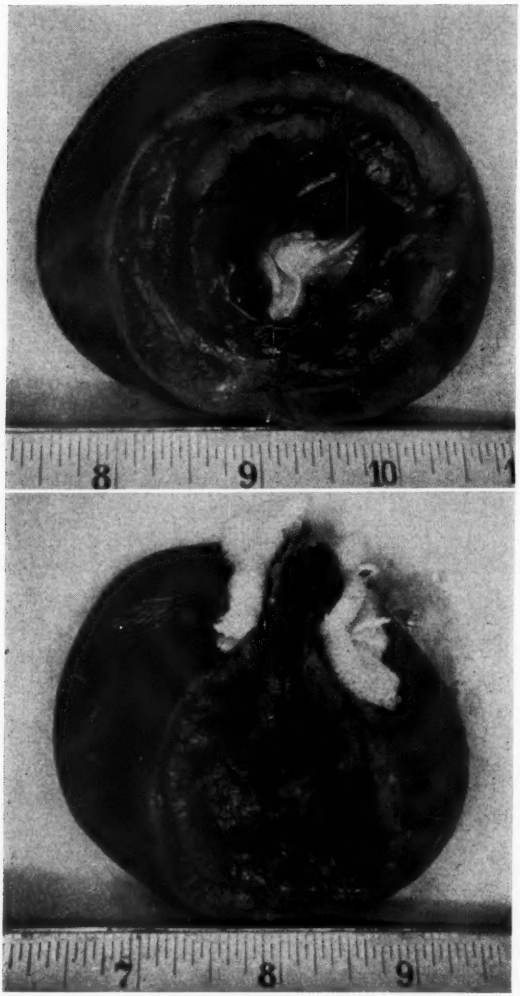
All of the dogs had one half or more of the anterior surface of the left ventricle infarcted. This was represented by a triangular area which on excision measured 3 by 3 by 3 cm. or more. A rim of 1.5 to 1.0 cm. of tissue surrounding this area was incorporated within the suture line and thus was excluded from the functioning myocardial musculature. Six of the dogs died

A.



B.

Fig. 13.—*A*, Cross-sectional view of the normal dog heart taken one third of the distance from the apex of the ventricle toward the base.

B, Similar section of heart of dog subjected to excision of an experimentally created infarct 13 days previously. Note the marked reduction in the size of the left ventricular chamber.

immediately from irreversible ventricular fibrillation. The hearts of two additional dogs fibrillated, but a normal sinus rhythm was re-established by electric countershock. Two dogs died of bleeding when the sutures were not overlapped properly. One died of empyema. Thirteen dogs died from the first to the ninth day with congestive heart failure. They exhibited pulmonary edema, engorgement of the liver, hydrothorax, and ascites. Three dogs were sacrificed on the tenth, thirteenth, and twenty-second days. They had similar findings. The longest lived dog in the series had massive ascites and hydrothorax and was deteriorating when sacrificed.

It had been noticed that immediately after the clamp was closed, there was a moderate to marked dilatation of the left auricle, the right ventricle, and the pulmonary artery. There was no immediate drop in blood pressure after the heart was infarcted[55, 56] but a gradual drop of 10 to 30 mm. Hg was observed after the clamp was applied.

The hearts were studied at post mortem. The inside of the heart showed only a narrow linear scar representing the site of excision. There was no evidence of mural thrombosis. On the outside, there were dense pericardial adhesions to the resected area, giving it a smooth surface. The suture line was represented by a tough fibrous scar. In the dog that was sacrificed at 22 days, the Ivalon sponge was well infiltrated with fibrous tissue. In no instance was a papillary muscle destroyed. When it was infringed upon it was caught up within the suture line. At no time was there evidence of mitral insufficiency as a result.

These hearts did not appear to show any compensatory dilatation to compensate for the excision of ventricular tissue. There was a reduction in the ventricular volume which appeared to be about half of its usual capacity (Fig. 13, *A* and *B*). These findings were consistent in all the surviving dogs.

DISCUSSION

The net conclusion from this work is that the heart is not capable of free and unrestrained dilatation and that this cannot be expected promptly after surgical reduction in the size of the left ventricle, significantly below normal limits. Instead there is marked restriction in the stroke and total cardiac output and a type of circulatory obstruction similar to that observed in experimentally produced mitral stenosis.

While the considerable increase in left ventricular volume which is characteristic of hearts with myocardial aneurysm would seem to militate against easy production of such functional inlet obstruction, it is evident that there must be a limit even in these cases to the amount of permissible ventricular wall resection. After all, the predominant source of such aneurysms is the very large infarction which is caused by occlusion of a major coronary arterial branch comparable to that ligated in the experimental animals. Since the normal process of healing of an area of myocardial infarction is by fibrous replacement, it is clear from the usual benign course in uncomplicated clinical cases that the presence of such areas of scarring, per se, are not deleterious

to heart function. Their presence does not necessarily imply an enlargement of the ventricular lumen since they merely may replace an equal area of destroyed muscle tissue.

Since the aneurysmal sac or pouch characteristically represents but the thinned-out central portion or fundus of the postinfarctional scar, it would seem logical merely to amputate this bulging area and to use the remaining peripheral portion of the scar for closure of the created ventricular wall defect. The combined fibromuscular structure of this peripheral portion of the original area of healing represents a nearly ideal tissue for suturing, being both strong and supplied with a rich capillary type of vasculature (augmentation of the intercoronary artery communications).

We, therefore, conceive it to be the surgeon's responsibility in a case of ventricular aneurysm to remove such portion of the postinfarctional scar as is necessary to overcome the deleterious physiologic effects of the aneurysm and, at the same time, to preserve sufficient of its periphery to reconstruct at least a minimally adequate ventricular chamber. In other words, he must "tailor" the ventricular cavity to as near the normal size and shape as is feasible. Such concepts are implicit in the term "ventriculoplasty."

We believe that excessive removal of the scarred portion of the ventricle may have been responsible for the development of persistent heart failure in some of the reported late deaths following excision of ventricular aneurysms.

It, also, is manifestly the surgeon's responsibility to avoid inadvertent dislodgment of clotted material which may be contained within the aneurysmal pouch during the manipulations incident to its removal. While this might be accomplished by the use of an open technique and a heart-lung bypass, we feel that it may be done more simply and with equal effectiveness by the use of the "flush-out" technique as described.

ILLUSTRATIVE CASE REPORTS

CASE 4.—V. F. (Hospital No. 54-04387), a 56-year-old white male Brazilian coffee planter, was admitted to the Hahnemann Hospital in Philadelphia on April 8, 1954, with a history of a myocardial infarction in January, 1953. In May of 1953, he had ventricular failure and at this time a diagnosis of left ventricular aneurysm was made. This was controlled by medical therapy and the patient did moderately well until December, 1953, when he again went into failure which persisted in mild form in spite of vigorous medical therapy.

On admission to the hospital, the patient was dyspneic at rest and orthopneic. The blood pressure was 124/80 mm. Hg. A diastolic gallop rhythm was present. The heart sounds were distant but no murmurs were heard. There were moist râles at both bases, the liver was palpable three fingers below the costal margin, and there was a one plus ankle edema. The electrocardiogram showed evidences of an old anterior wall infarction. The roentgenogram demonstrated a bulging on the anterolateral surface of the left ventricle. A diagnosis of an old anterolateral infarction with a ventricular aneurysm was reached and the patient was prepared for surgery. In spite of digitalis, bed rest, mercurial diuresis, and a salt-poor diet the gallop rhythm and dyspnea persisted.

On April 15, 1954, a left posterolateral thoracotomy was performed. An anterolateral infarction was found, and a ventriculoplasty was performed (Fig. 14). The patient tolerated the procedure well and left the operating room in good condition. The diastolic gallop disappeared on the first postoperative day, was heard intermittently during the first week, and then disappeared.

The patient was kept at bed rest for 5 weeks and then observed for 7 weeks. During the latter period he was able to walk and to climb three flights of stairs without dyspnea.

The patient was re-admitted to the hospital for check up in December, 1956. His only complaint at this time was of occasional bouts of deep and fast breathing which occurred during sleep, causing him to awaken.

He had no evidence of failure or of further cardiac deterioration. However, he was still maintained on digitalis.

Comment: This man, who was, historically, the first patient to survive ventricular resection for cardiac aneurysm,[13, 34, 56] was practically terminal at the time of surgery. Yet his operative response was gratifying and he remains well to date. At check up 2½ years after resection of the ventricular aneurysm, he seemed to be following the usual relatively satisfactory course to be expected of a patient who had suffered a myocardial infarction without complication. He continues to do well 3 years following operation (Fig. 15, *A-D*).

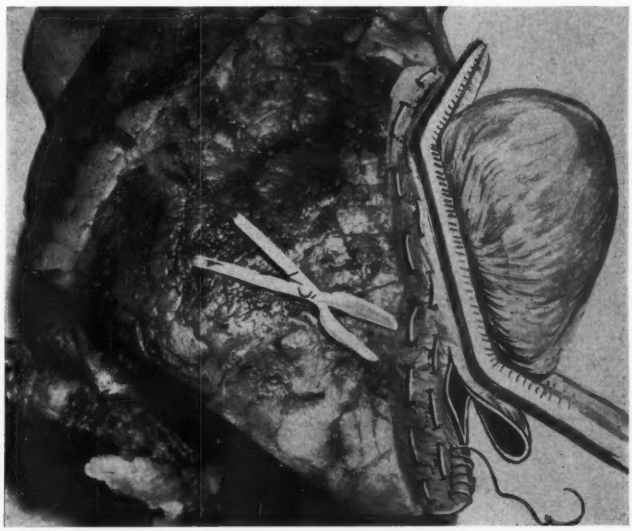


Fig. 14.—Semidiagrammatic representation of technique of ventricular excision employed in V. F. (Bailey, Surgery of the Heart, ed. 1, Philadelphia, 1955, Lea & Febiger, p. 1045.)

CASE 5.—R. T. (Hospital No. 56-19642), was a 56-year-old white woman who, on April 30, 1955, had a sudden onset of severe chest pain. This was diagnosed as a myocardial infarction and the patient was hospitalized for 2 months. During July, 1955, enlargement of the cardiac silhouette was noted on x-ray studies and the patient was placed on digitalis therapy. In December, 1955, a bout of atrial fibrillation occurred, accompanied by ankle edema and shortness of breath. This was treated with quinidine. She improved somewhat during the next few months but still had marked limitation of activity because of shortness of breath. The patient was admitted to the Hahnemann Hospital for evaluation.

There was no neck vein distention, the lungs were clear to auscultation and percussion. The heart exhibited a normal sinus rhythm, the sounds were diminished in intensity, and there were no murmurs. The blood pressure was 104/60 mm. Hg. The liver was not palpable and there was no ankle edema.

The electrocardiogram showed findings indicative of a previous anterolateral myocardial infarction. Routine x-ray studies showed a bulging and elevation of the superior portion of the left ventricle, with a discontinuity of its border. Cardioangiography, by left atrial puncture, demonstrated a large ventricular aneurysm (Fig. 16, *A* and *B*).

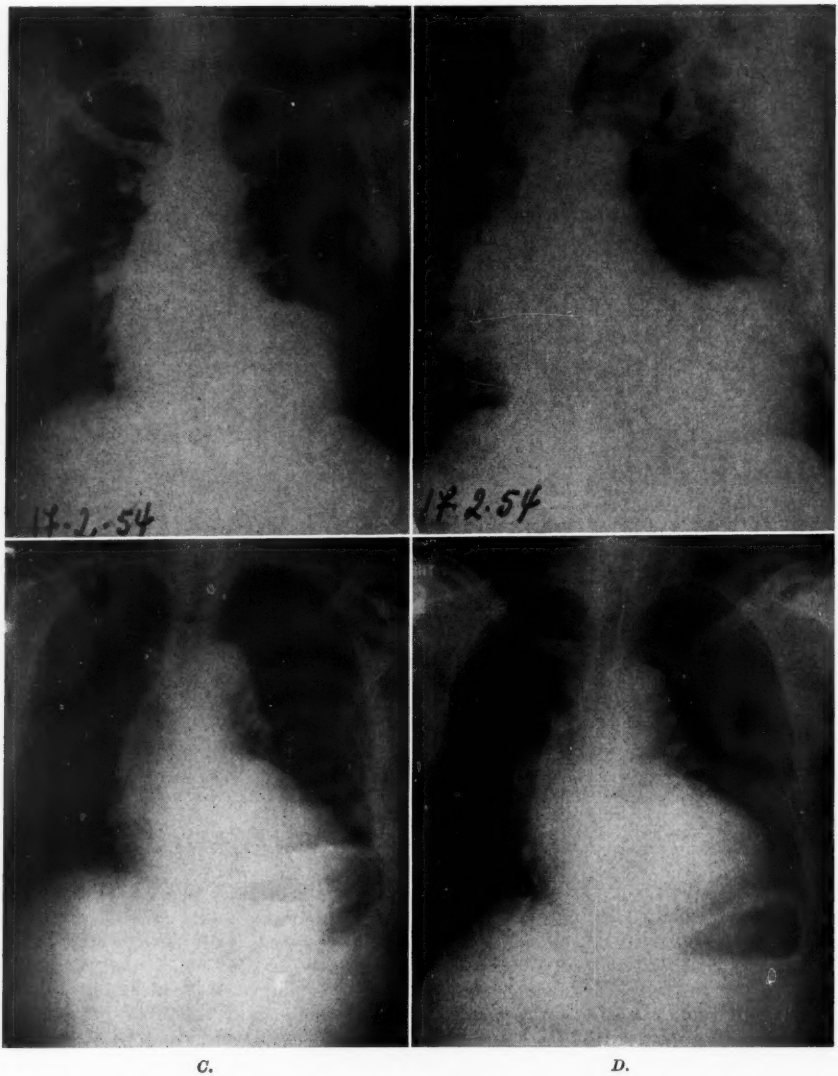
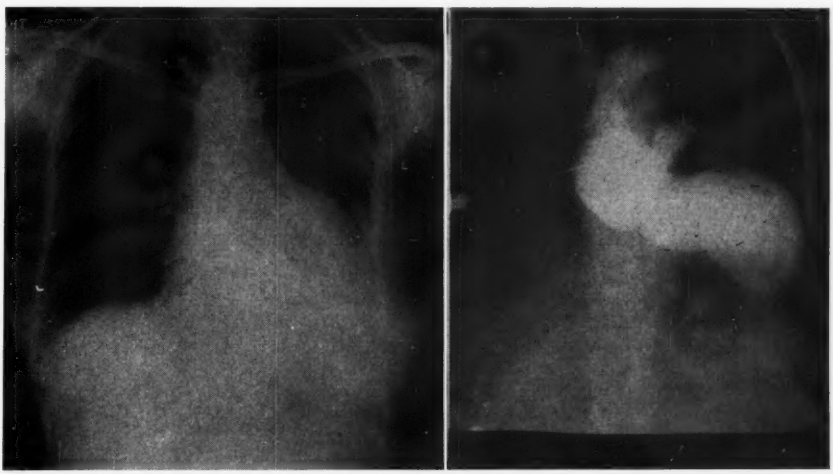


Fig. 15.—*A* and *B*, Preoperative PA and oblique roentgenograms of V. F. (Case 4) showing a bulging of the left border of the heart which was diagnosed as a postinfarctional ventricular aneurysm. (Bailey, Surgery of the Heart, ed. 1, Philadelphia, 1955, Lea & Febiger, p. 946.)

C, PA roentgenogram of the heart of V. F., 6 weeks after ventriculoplasty.

D, Comparable roentgenographic study of V. F. taken 2½ years postoperatively.

The patient was prepared medically and operated upon on April 30, 1956. After the induction of anesthesia it was noted that the carotid pulse was absent. The left chest was opened quickly and the heart was found to be in standstill. Cardiac massage was instituted and prompt restitution of the heartbeat was achieved with restoration of the blood pressure to a systolic level of 110. After recovery seemed assured, resection of the aneurysm was undertaken and a ventriculoplasty was performed. The patient tolerated the procedure well and left the operating room in good condition. The patient had an unremarkable convalescence and left the hospital on the seventeenth postoperative day. Her condition today is reported as significantly improved.



A. *B.*

Fig. 16.—*A*, PA roentgenogram of female R. T. (Case 5) showing abnormal cardiac silhouette.

B, Cardioangiogram obtained by left atrial injection showing large anterolateral aneurysmal shadow superimposed upon the shadow of the interior of the left ventricle (outline of latter indicated by dotted lines).

CASE 6.—R. T. (Hospital No. 56-28430), a 65-year-old white man, was admitted to Hahnemann Hospital on Dec. 2, 1956. In March, 1955, the patient had had an acute myocardial infarction for which he was hospitalized. Prior to this episode he had had some substernal discomfort not definitely associated with exertion. Following the infarction, he developed easy fatigability and dyspnea on exertion.

On admission the blood pressure was 120/70 mm. Hg. The lungs were clear to auscultation and percussion. The heart sounds were distant. No murmurs were discernible. The liver was not palpable and no ankle edema was present. The electrocardiogram showed changes indicative of extreme myocardial damage involving the anteroseptal portion of the left ventricle. Routine roentgenograms showed a bulging of the left ventricular wall which was believed to represent an aneurysm (Fig. 17, *A*). Ventriculography clearly demonstrated a large left ventricular aneurysm with a double pouch (Fig. 17, *B*). The patient was prepared for surgery with digitalis, Thiomerin, and bed rest.

At thoracotomy, on Dec. 17, 1956, two bulges were found adjacent to each other. The first was on the lateral aspect of the heart. The second was adjacent to the first in the region of the apex. Using the technique of ventriculoplasty, both aneurysmal bulges were resected. The patient tolerated the procedure well.

Postoperatively, the patient did well until the third day when he had a brief hypotensive episode which required a continuous infusion of norepinephrine. Subsequently, 650 c.c. of sanguineous fluid was aspirated from the left chest. No further difficulties occurred. A

postoperative ventriculogram revealed a nearly normal shaped left ventricle (Fig. 17, *C*). The patient was discharged 4 weeks postoperatively in good condition. A recent medical report is most satisfying, both dyspnea and fatigability being relieved.

A.

B. *C.*

Fig. 17.—*A*, PA roentgenogram of male R. T. (Case 6) showing aneurysmal bulge upon the anterolateral aspect of the left ventricle.

B, Preoperative ventriculogram (by direct ventricular injection) obtained from Case 6 showing double aneurysmal bulge.

C, Postoperative ventriculogram in Case 6 showing effect of ventriculoplasty upon left ventricular chamber.

TECHNIQUE OF VENTRICULOPLASTY

Depending upon the site of localization of the aneurysmal dilatation as determined by fluoroscopy and cardioangiographic opacification, the operative

approach is made through a left anterolateral intercostal (fourth or fifth) incision for anteriorly or anterolaterally located aneurysms, or through a left posterolateral one for a lateral, posterolateral, or diaphragmatic projection, in this case using the fifth, sixth, or seventh intercostal space (whichever seems most appropriate). After the pericardium is exposed, it may be incised and dissected away from the aneurysmal bulge, the integrity of the left phrenic nerve being preserved.

When the aneurysm has been exposed as completely as possible, the manner in which the ventricle is to be tailored is determined. In most cases, this will amount to simple longitudinal application of the Rumel-Morse clamp to the basal portion of the aneurysm in such a way that after the fundus of the aneurysmal bulge has been amputated the contour of the repaired heart wall will be restored as nearly as possible to normal. Careful inspection and palpation of the surface of the heart will indicate the possibility or likelihood of dividing or occluding by the suture line any sizable functioning coronary arterial branches, and also will disclose any area of unusual thinning of the ventricular wall which would be retained should the proposed line of resection fail to include it.

The operator's ungloved right index finger is inserted into the left atrium by way of an incision made into the purse-stringed tip of the left auricular appendage. At transient strategic intervals the finger tip is advanced through the (normal) mitral valve to explore the interior of the left ventricular cavity and perhaps that of the aneurysm itself. The localization of the sites of insertion of the papillary muscles with respect to the aneurysm is investigated. If excessive or loosely attached thrombotic material is present, suggesting the danger of partial dislodgment at the moment of clamping of the base of the aneurysm, especial care is taken with the steps of the "flush-out" technique.

Having determined the extent and location of the portion of the scar to be resected, the special Rumel-Morse clamp is applied with a safe margin so that the long teeth upon its opened jaws penetrate deeply into the partly fibrosed ventricular wall. After everything is in readiness, blood transfusion having been administered to a slight excess, the portion of the aneurysm to be resected is suddenly and widely incised, permitting a momentary massive gush of blood from the ventricular cavity which tends to flush out any loosely attached clot within the sac. Then, the jaws of the clamp are approximated, abolishing further blood loss and restoring the functional integrity of the circulation (Fig. 18, *A-D*). If necessary, further transfusion of whole blood is administered.

The selected portion of the now excluded fibrosed ventricular wall is excised. Any visible, partially adherent, thrombotic material may be extracted from between the protruding ventricular lips. These are approximated with a row of interrupted interlocking mattress sutures of heavy silk placed either above or below the clamp at a distance of 8 to 10 mm. from the cut margin. The cut edges are oversewn with a continuous suture of medium silk. In cases or in areas where there has not been sufficient fibrous infiltration of the residual wall to render it tough and strong, the suturing needles may be passed through

strips of compressed plastic sponge,* 2 cm. wide and 3 mm. thick. This material acts as an excellent buffer between the epicardium and the suture loops which otherwise might tend to cut through.

After the line of repair has been completed, the clamp is released a little at a time until the two rows of sutures are restraining the full intraventricular pressure. If any bleeding occurs from the suture line, one or more additional mattress stitches may be placed to control it, preferably without re-application of the clamp.

Fig. 18.—*A*, Rumel-Morse fenestrated and toothed ventricular clamp. (Surg., Gynec. & Obst. 104: 539-542, 1957.)

If enough pericardial tissue remains to cover the site of ventricular repair, it may be so utilized. However, an adequate and dependent pericardial opening must be provided to permit free drainage of serosanguineous exudate into the left pleural space. The latter is drained into a water-seal bottle by a multiwindowed No. 28 Fr. urethral catheter inserted through a stab wound made in the seventh or eight intercostal space in the posterior axillary line.

The thoracic wall is repaired in layers, using pericostal and fascial sutures of No. 2 chromicized catgut. The skin is closed with continuous running or subcuticular stainless steel wire.

The method of repair of a true ventricular aneurysm by an open technique, as suggested by Lillihei,[57] does not differ greatly from this except for the necessity for cannulation and the operation of the cardiopulmonary apparatus. Complete circulatory bypass is preferable to merely a left-sided one. With this technique, the special Rumel-Morse clamp is not necessary, the

*Ivalon: Obtainable from Davis and Geck, Danbury, Conn.

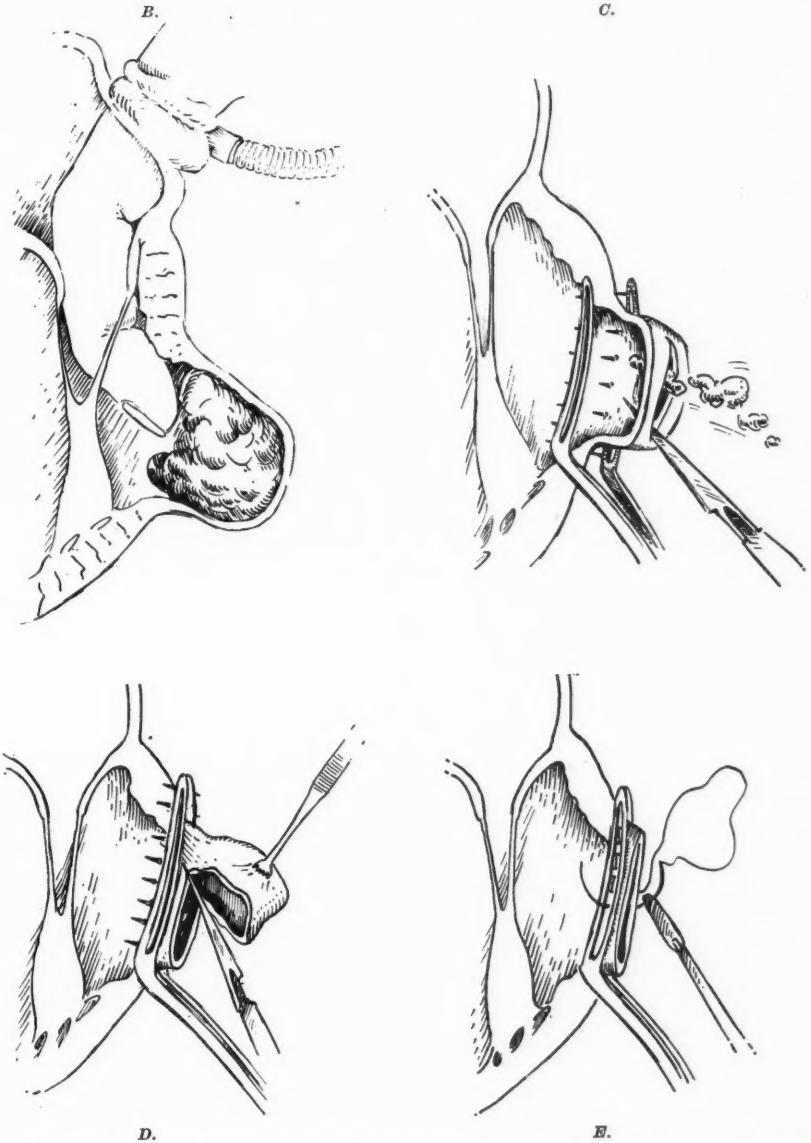


Fig. 18 Cont'd.—*B*, Operator's ungloved right index finger is inserted into the left ventricle by way of the left auricular appendage and the mitral valve to explore the chamber and to detect the presence of any loose or excessive thrombotic material.

C, The teeth of the clamp are applied securely along the selected line and the fundus of the unclamped aneurysm is incised widely to "flush out" any loosely attached thrombotic material.

D, The jaws of the clamp are closed to restrain bleeding and the excluded aneurysmal wall is trimmed away.

E, The ventricular lips are approximated by mattress sutures applied through the slots in the fenestrated jaws of the clamp. After removal of the clamp, the cut edges are oversewn. (Bailey and Gilman, Surg., Gynec. and Obst. 104: 539-542, 1957. Courtesy of the Franklin H. Martin Memorial Foundation.)

selected portion of the bulging ventricle simply being excised and the interior of the chamber being exposed. Any clots which extend into the left ventricle are removed by suction or extracted mechanically.

After displacing the air from the open left ventricular (and atrial) chamber by gaseous carbon dioxide (in order to obviate the possibility of air embolization), the cavity is filled with blood or saline, and the heart wall is repaired in a manner similar to that described for the closed method.

SUMMARY AND CONCLUSIONS

With the increased incidence of surgical ventriculotomy, we may expect in the future to encounter more ventricular aneurysms of traumatic (false) type. While every effort should be made to prevent their development, we feel that they may be corrected with relative safety by the technique herein described.

Since adopting the "flush-out" technique, following the death of our second postinfarctional patient from embolization, and by continuing the use of the principle of ventriculoplasty (tailoring the ventricle) rather than attempting totally to excise all scarred tissue, there has been a gratifying surgical and clinical response from each patient so operated. In view of the fact that no "revascularization procedure" was included in the operative technique, it is believed that all observed benefit was due to the removal of the physiologic burden imposed by the paradoxically pulsating sac itself. We *feel* that a widespread application of these concepts and techniques will enable others to achieve equally good results.

In view of the gloomy outlook for individuals with symptom-producing postinfarctional ventricular aneurysm, and the rather dramatic response to appropriate operative intervention, we strongly urge surgical consideration for these common, although less often recognized, sequelae of coronary arterial disease. Knowledge that an effective surgical method of treatment exists no doubt will stimulate an increased awareness on the part of the internist that such a lesion may exist. By appropriate opacification techniques the diagnosis may be established with certainty and the size, shape, and localization of the aneurysm determined.

It is to be hoped, in the future, that these patients may be referred for surgery at a somewhat earlier stage in their devolutionary course when the chances for survival and clinical "cure" will be greater, rather than in the near-terminal or pre-terminal state in which we have seen them to date.

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Discussion

DR. EGBERT H. FELL, Chicago, Ill.—At the Cook County Hospital, Dr. B. M. Gasul and his associations had an opportunity of studying a 7-month-old, healthy looking infant who had a loud machinery murmur at the level of the cardiac apex.

(slide) The angiocardiogram of the right side of the heart is normal, but on the left side of the heart we see a very large right coronary artery filled with dye ending in an aneurysmal-like swelling at the apex of the heart.

(movie) We have had one other patient with an anomalous coronary artery entering the right ventricle just proximal to the pulmonary valve with no aneurysmal dilatation, and a preoperative diagnosis of patent ductus arteriosus. Here we have a true arteriovenous aneurysm at the apex of the heart; the movie shows the aneurysm very well, and you see the large right coronary artery coming up from behind. The coronary artery was clamped, divided, and sutured. Blood now enters the aneurysmal sac from the right ventricle. The coronary artery was every bit of four times its normal size. The wall of the aneurysm was resected and the ostia are seen, one from the coronary and one from the right ventricle. The ventricular defect was closed in two layers. Pathologic studies of the resected material confirmed it to be ventricular wall. The child is now about 2 months postoperative and is doing well.

This movie has shown an unusual experience we have had at the Cook County Hospital with a congenital arteriovenous fistula of the right coronary artery and right ventricle with an aneurysm of the apex of the right ventricle. It was diagnosed prior to surgery and treatment resulted happily in a cure.

DR. J. L. MANGIARDI, Garden City, N. Y.—I wish to congratulate Dr. Bailey for his excellent results in pioneering a new operation and to add a case report.

(slide) This is one of the 25,000 cases available in 1956 that Dr. Bailey mentioned. This 44-year-old man presented with paradoxical pulsations over the aneurysm and severe hypertension ranging from 150 to 250 systolic. He had no history of coronary artery occlusion, but the electrocardiogram was compatible with an anterior wall infarction and aneurysm.

(slide) This is a slide taken 2 years prior to admission and shows no evidence of aneurysm at that time so that, with the electrocardiogram, we felt quite confident of our diagnosis of postinfarction myocardial aneurysm.

(slide) We had just read Dr. Bailey's original report of his first successful case, and decided to operate on this patient. This shows technique of resection—but it is not absolutely correct, we actually had three suture lines, the first a continuous mattress suture, the second an interrupted layer, and the third sutured over and over with fine silk exactly as shown in the previous slide. Before resection, I might mention that we thought the aneurysm was full of clot and we had in mind to stab the apex, allow clot to extrude, and then clamp the base. However, we were very fearful that our reflexes would not measure up to Dr. Bailey's criteria, so we did not do it. The base was simply cross-clamped, and although an embolus was thrown off, it was fortunately very small, as the patient recovered rapidly and is now well in all respects, a year and a half postoperatively.

(slide) Here is the specimen. This *is* a centimeter rule. Looking at the aneurysm from within its cavity, you can see a large amount of clot and fibrin; there was more on the specimen at the time this was taken off.

I was a little surprised at what Dr. Bailey had to say about muscle in the wall of the aneurysm. We were surprised and very disappointed because we did not find muscle in the wall of this resected area.

(slide) This shows the microscopic appearance of the aneurysm. It has been of great interest to us that this man's hypertension has never risen above 160 ever since the time of resection of this aneurysm. So far as the hypertension is concerned, the only thing I can think of is the decrease in size of the left ventricular cavity, but of course this remains a question.

(slide) We have had a lot of cardiographs in the follow-up of this case, and our cardiologist is much interested in their comparison. This is the immediate preoperative study, and I think you can see the high S-T segments in Leads I and AVL particularly, and inverted T waves.

(slide) This is an immediate postoperative cardiogram. The S-T segments are now normal, and the T waves are flat. Very little change has occurred since.

DR. LEWIS H. BOSHER, JR., Richmond, Va.—We have encountered still another type of myocardial aneurysm which because of its rarity is perhaps worth recording here. This was the case of a 33-year-old Negro woman who entered the hospital in chronic cardiac tamponade. She had an elevated venous pressure of more than 250 mm. of saline, a depressed blood pressure and pulse pressure, and a very large liver. The cardiac silhouette was quite large; aspirations of the pericardium were done which yielded almost frank blood. This temporarily relieved the cardiac tamponade. Because of the rather marked abnormality of the clotting mechanism, operation was temporarily deferred. She had a very low fibrinogen, and this was corrected by intravenous fibrinogen. At operation several days later, this was encountered:

(slide) The pericardial sac was markedly enlarged and had a large quantity of bloody fluid present in it. On the wall of the right atrium very close to the atrioventricular groove there was a saccular, paper-thin aneurysmal dilatation about 4 cm. in diameter. The atrium was explored as we thought that perhaps some type of intracardiac tumor was present, but none was found. The wall of the right atrium itself seemed somewhat dilated, perhaps due to tricuspid insufficiency. This was not definitely established. The wall was quite friable next to this sacculation.

(slide) At the top of the slide you see the paper-thin aneurysm which bled on contact with the fingers and which sealed spontaneously.

(slide) A slab of polyvinyl sponge was sutured down over the aneurysm and this concluded the operative procedure. No cardia was found in the pericardial fluid and thus this represents a mycotic aneurysm. This patient's low-grade fever did not subside postoperatively until after an intensive course of therapy with sulfadiazine. She remains well 6 months later.

DR. JOHN C. JONES, Los Angeles, Calif.—I enjoyed Dr. Bailey's discussion and I merely arise to tell you that I came unprepared to discuss my one case. But I bring up this one point. This case of mine was operated upon before Dr. Bailey published his technique, and I do not believe that whether or not you anticipate there is a clot you should put a finger inside to release the clot before you get a chance to resect the aneurysm. The other point is that I do not believe the best technique is to put a clamp on these ventricles. In the first place you will probably ruin your clamp, and in the second place it is not good technique anyway.

I think as we confront this problem, the best way to resect an aneurysm is to place the sutures first, resect the aneurysm, and then draw up the sutures and tie them, and then you will have no difficulty; you have a clot that will come out, if you do not have a clot you are not in trouble, and if you put your finger inside you are likely to release the clot.

The patient I operated upon was in severe decompensation, he had been decompensated for years, and he is now particularly well compensated and has done very well. Just the simple manipulation of placing the sutures, resecting the aneurysm, and tying them, was effective.

DR. DENTON A. COOLEY, Houston, Texas.—I think from the standpoint of completeness it may be worth while to state that there are other complications of coronary occlusion and myocardial infarction which at this time may be surgically correctible. I refer to the acutely perforated ventricular septum and to the ruptured papillary muscle. Both of these are highly fatal complications of coronary disease but, with the methods of cardiopulmonary bypass at our disposal, they could be handled successfully in some instances. We have had experience with one patient with perforated ventricular septum who went through the operation satisfactorily. Unfortunately he died 6 weeks later of pericarditis and empyema. We have operated in 2 cases of ruptured papillary muscle. Neither of these were secondary to myocardial infarction; one being due to trauma, the other a complication of bacterial endocarditis. In both of these patients the papillary muscle could be repaired. Perhaps in the future, cardiac surgeons may adopt a more aggressive attitude toward these complications of acute myocardial infarction.

DR. ALFRED GOLDMAN, Beverly Hills, Calif.—I rise only to mention another type of ventricular aneurysm; that is one in which one cannot get around its base. We have had two such cases, and each time an attempt was made to clamp this very leathery dilated wall of the left ventricle, the blood pressure would drop. In each of these cases we made a repair after Beck's method, using a flap of pericardial fat pad and pericardium over the small weak spots that were encountered on the surface of the aneurysm. Both these patients are now about 2 years postoperative and neither has had any further coronary occlusion. They are both somewhat disabled cardiac-wise, but they are both alive and able to do light work.

I want to mention one test that might be valuable in determining the extent of a through-and-through infarct such as is present with this type of aneurysmal dilatation of the left ventricle. If the surface of the heart is explored with the unipolar electrode, using Prinzmental's technique there will be over any active myocardium, with pressure, an injury current measured by an elevation of the S-T segment. Over the surface of this kind of ventricular aneurysm, where there is no active muscle, Q waves occur without elevation of S-T segment on pressure with the electrode. One can demarcate in this way the edges of the aneurysmal, dilatation because pressure will produce no rise of the S-T segment over the through-and-through infarct replaced by fibrous tissue.

DR. CLAUDE S. BECK, Cleveland, Ohio.—Not all aneurysms of the heart are saccular, and not all of them need to be excised. I have had 2 patients with aneurysm large enough to have a paradoxical pulsation. These were approximately 4 cm. in diameter and bulged out approximately 2 cm. A graft of fascia lata was placed over the aneurysm in one and in the other a graft of nylon was used. These grafts were sutured to the adherent pericardium in the periphery of the aneurysm. The graft was made tight enough to do away with the paradoxical pulsation and the outward bulge. This method of treatment is recommended for aneurysms with a paradoxical pulsation and without a narrow neck. In patients upon whom I operated for coronary artery disease, the incidence of aneurysm was 7 per cent. The only aneurysms that need a supporting graft are those that definitely bulge out from the heart or have a paradoxical pulsation.

DR. CHARLES P. BAILEY, Philadelphia, Pa. (Closing).—I want to thank all the discussors and I will try to answer the questions they have brought out. There is little to say about Dr. Fell's beautiful case, and we can only congratulate him upon his success. Dr. Mangiardi raised the question of why there was no remaining muscle tissue in the section of the aneurysmal wall. That is, of course, the end result of an infarction that was through-and-through. Some aneurysms do have muscle fibers in their wall, and they are the ones that occur after incomplete or partial thickness involvements. Those that occur after secondary infarction of a previously infarcted area tend to be purely fibrous. I do not know why Dr. Mangiardi's patient lost his hypertension, but it would be a wonderful thing if we could only control this effect upon our patients.

It is important to realize that even though there be muscular elements in the wall of an aneurysmal pouch, as long as you do not get beyond the limits or confines of the scar you will have good strong tissue to hold the sutures, something you do not have in the experimental animal. Therefore in the human patient you will seldom need Ivalon sponge reinforcement of your suture line.

Dr. Bosher's case is indeed fascinating; I suppose none of us has ever heard of anything like that before, and he has had a fine result. Dr. Jones, I think, is somewhat more fearful than he need be about clots within the aneurysm. If you are gentle you can palpate the clot and not dislodge it. I think it is important to know whether or not one of the papillary muscles is involved in the aneurysm, because if you are going to cut away its normal site of attachment you must be particularly careful to incorporate the stump within the suture line in the ventricular wall, lest you produce a severe degree of mitral insufficiency. I think there may be many other ways than ours of taking off an aneurysm and I have no doubt Dr. Jones' method will work. In fact, about a year prior to our first case, Dr. Glover, then my associate, did remove a ventricular aneurysm using a purse-string suture technique. The patient did not survive, but this probably was not related to the technique; there was an abdominal complication. We think that if Dr. Jones would get one of these special clamps which are available from the Pilling Company, he would not have to worry about breaking it.

Dr. Cooley's fascinating case of repair of the acutely perforated ventricular septum, of course, clearly shows the surgical possibilities in chronic aneurysm of the septum. That is not a very common form of ventricular aneurysm but it does occur, and I cannot imagine any way of treating it except by the use of cardiopulmonary bypass and an open technique. As a matter of fact, Dr. Lillehei has already removed a ventricular aneurysm by an open technique.

CLINICAL EVALUATION OF THE SURGICAL TREATMENT OF THIRTY-TWO CASES OF CORONARY ARTERY DISEASE

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THE ultimate objective in the approach to the problem of arteriosclerosis of the coronary arteries is prevention. Since a reliable method of prevention has not yet been developed, attention must be directed toward overcoming the effects of occlusive narrowing of the coronary arteries. Surgical revascularization of the heart provides a means of improving coronary circulation.

Schlesinger and Zoll[1] have shown that only the larger coronary arteries are affected by arteriosclerosis. The smaller arteries, and the veins and capillaries are not involved. These vessels, therefore, do not offer an obstruction to coronary blood flow. If oxygenated blood could be delivered to the uninvolved vessels, and the blood were equitably distributed throughout the myocardium, it is conceivable that the effects of diminished coronary inflow could be overcome. The validity of this theory has been demonstrated in the animal laboratory. Beck has shown that the mortality and the size of an infarct resulting from sudden occlusion of a major coronary artery can be significantly reduced in dogs following the Beck I[2] and Beck II[3] operations. These operations also produce a significant increase in coronary backflow as demonstrated by the Mautz-Gregg backflow studies.[4]

The results of these experiments have led to the development of certain principles required of a successful revascularization procedure. These principles include: (1) the delivery of an additional quantity of oxygenated blood to the ischemic areas of the heart, (2) the utilization of existing anastomotic channels, and (3) the stimulation of new intercoronary communications to provide a more equitable distribution of oxygenated blood.

Unfortunately, translation of laboratory data to the human being cannot be done with certainty. Objective demonstration of the value of a revascularization procedure cannot be obtained in the human being at this time. Results, therefore, must be evaluated on the basis of (1) amelioration of symptoms, (2) ability to return to work, and (3) length of survival. Proof of the feasibility of surgical revascularization of the heart must depend upon evaluation of a large number of cases closely observed for a long period of time.

Received for publication Jan. 16, 1957.
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SELECTION OF CASES FOR SURGERY

As an aid in the selection of cases for surgery the status of the patient with coronary artery disease was classified according to the following categories.

1. Asymptomatic: Those patients who have had previous myocardial infarction but who no longer have symptoms. It was assumed that collateral circulation developed to such a degree as to overcome the effects of coronary occlusion and surgery was not indicated.

2. Progressive: Those cases in which the collateral circulation is unable to keep pace with a rapidly advancing disease process. Existing collateral circulation was considered to be inadequate for the patient to withstand surgery.

3. Stabilized: Patients with angina pectoris that is stabilized with regard to frequency and severity of attacks. This type of individual represents the ideal candidate for surgery.

4. Far-Advanced: Those patients who have severe coronary artery disease with advanced myocardial degeneration and replacement fibrosis. Obviously, surgery can be of little value in this group since revascularization cannot alter the irreversible myocardial changes.

In the more specific selection of cases for surgery the following considerations arose.

1. Age: Age per se was not a factor in the selection of cases for surgery except for the Beck II operation and combined procedures. Physiologic age was considered to be of greater importance than chronological age.

2. Sex: Sex was not an important factor in the selection of candidates for surgery.

3. Time of Surgery: Surgery was not performed for at least six months after coronary occlusion. This time interval was fixed arbitrarily to ascertain the degree of anastomotic circulation that may have developed after infarction. If sufficient collateral circulation had developed, surgery was not deemed necessary.

4. Angina Pectoris: Angina of effort represented the prime indication for surgery. It is important to emphasize that the angina had to be stabilized with regard to frequency and severity of attacks. Status anginosus and angina decubitus contraindicated surgery.

5. Hypertension: Patients with severe degrees of hypertension were not accepted for surgery. A mild or moderate elevation of blood pressure without evidence of systemic degeneration was not considered a contraindication to surgery. Blood pressure was an important factor in the selection of patients for the Beck II operation and combined procedures.

6. Heart Size and Congestive Failure: The ideal candidate should not have evidence of cardiac enlargement or congestive failure. A slight degree of enlargement and mild congestive failure were acceptable. Patients with

more than minimal cardiac enlargement and evidence of persistent or progressive cardiac decompensation have advanced myocardial degeneration and fibrosis and cannot be aided by revascularization procedures.

7. Disturbances in Rhythm: Surgery was considered to be hazardous in the presence of arrhythmias since they are generally associated with extensive myocardial damage.

8. Associated Conditions: Advanced medical diseases such as generalized arteriosclerosis, pulmonary disease, hepatic or renal failure contraindicated surgery.

PREOPERATIVE AND POSTOPERATIVE CARE

All patients selected for surgery were admitted to the hospital one week prior to operation. Laboratory examinations included a standard 12-lead electrocardiogram, x-ray and fluoroscopic examination of the chest in the posteroanterior, left anterior oblique and right anterior oblique views, complete blood count, sedimentation rate, nonprotein nitrogen, urinalysis, and serologic test for syphilis. It has been our practice in the past three years to digitalize completely all patients routinely prior to surgery. Mercurial diuretics were administered if there was any suspicion of cardiac failure.

Postoperatively, the patient was placed in an oxygen tent for twenty-four hours. A pleural drainage tube was attached to a water seal and removed twenty-four hours after drainage stopped. Digitalis was continued postoperatively. Bronchoscopic examination and removal of bronchial secretions were performed as necessary. An electrocardiogram was obtained on the second postoperative day and at the time of discharge. A portable film of the chest was taken on the first postoperative day and films comparable with preoperative x-rays were obtained prior to discharge from the hospital. Narcotics were administered for the relief of pain. Blood transfusions and intravenous fluids were used only when indicated. If necessary, blood pressure was maintained by the slow, continuous, intravenous drip of Levophed. Antibiotics were administered routinely, postoperatively. Skin sutures were removed on the tenth postoperative day at which time the patient was permitted out of bed. Physical activity was restricted for the first month and then increased gradually until the end of the second month when full activity was resumed. Digitalis was continued until the end of the second month postoperatively. Nitroglycerine, gr. $\frac{1}{400}$ every three hours, was administered during the first three postoperative months. The patient was permitted to return to work between the fourth and the sixth month.

RESULTS

This series includes 32 patients with coronary artery disease operated upon during the past five years (Table I).

Age.—The youngest patient was 32 and the oldest was 64 years of age at the time of surgery. The average age of the entire group was 44 years.

Sex.—Only 3 of the 32 patients were females. No attempt was made to exclude a patient from surgery solely on the basis of sex. The higher incidence of coronary artery disease in the male and his desire to seek rehabilitation were the chief reasons accounting for the sex differential.

Angina Pectoris.—All patients subjected to surgery had angina pectoris. Nineteen of the 32 patients had to discontinue employment. The remaining 13 patients had to change to employment which required less physical and emotional effort or continue their occupation despite varying degrees of disability. The determining factors in the selection of cases for surgery were the frequency and severity of anginal attacks, and the degree of incapacitation. Of the 32 patients, 28 had moderate to severe angina with attacks appearing on walking three city blocks or less. In the remaining cases it was less severe. In 18 of the 32 cases, angina had been present for two years or more.

Of the 29 patients who were followed up to five years postoperatively, 24 or 82.7 per cent had an excellent result. They are completely free of angina and have been able to return to their former occupations. In one of the remaining cases the patient continues to have angina, but of much less frequency and severity than before operation. He has been able to return to part-time work. There were 2 fair results. These occurred in patients who had an omentopexy operation in addition to the Beck I operation. Two patients had a poor result. One died eighteen months after a Vineberg operation. The other has had repeated angina and several episodes of myocardial infarction and is still unable to resume his former occupation.

The most dramatic results were obtained following a successful Beck II operation.

Previous Myocardial Infarction.—For the purpose of this study an attempt was made to accept for surgery only those patients who had definite evidence of coronary artery disease. Therefore, previous myocardial infarction was an important factor in the selection of patients for operation. A definite history of myocardial infarction was obtained in 28 of the 32 cases. Thirteen of the 28 patients had more than one infarction. Of the remaining ones, 2 had strong but not conclusive evidence of coronary occlusion. Two patients did not have a history of infarction.

No patient was operated on sooner than six months following coronary occlusion. The longest interval between infarction and surgery was seven years.

Cardiac Enlargement and Congestive Failure.—Definite left ventricular enlargement was present in 7 of the 32 cases. One of these patients had marked enlargement of the heart with an advanced degree of congestive failure. He died on the operating table. Of the remaining 6 patients, 5 had associated dyspnea on exertion. Five patients had exertional dyspnea without evidence of cardiac enlargement. One patient had paroxysmal nocturnal dyspnea. All patients were relieved of their dyspnea postoperatively, and in all cases the heart size returned to normal.

TABLE I. SUMMARY OF THE CLINICAL HISTORY AND OPERATIVE RESULTS OF

CASE NO.	NAME	SEX	AGE	PREOPERATIVE								
				ANGINA*		EMPLOY-MENT	DYSPNEA	CON-GESTIVE FAILURE	CARDIAC ENLARGE-MENT	ECG	NO. OF PREVIOUS MYO-CARDIAL IN-FARC-TIONS	
				THRESHOLD (BLOCKS)	DURATION							
BECK II												
1	J. C.	M	43	2	6 yr.	Discont.	Exertion	–	–	Old post.	3	
2	L. O.	M	56	½	2 yr.	Discont.	–	–	L. vent.	Old ant.	3	
3	J. T.	M	39	2	1 yr.	Discont.	–	–	–	Old post.	1	
4	W. K.	M	39	10	2 yr.	Cont.	–	–	–	Old post.	1	
5	W. L.	M	35	2	6 yr.	Cont.	Exertion	–	–	Old ant.-sept.	2	
FIRST STAGE BECK II												
6	W. S.	M	53	½	5 yr.	Discont.	Exertion	+	L. vent.	Right bundle branch block; old post.	2	
7	T. W.	M	38	2	6 mo.	Discont.	Nocturnal	–	–	Old post.	1	
BECK I PLUS OMENTOPEXY												
8	W. K.	M	45	½	6 mo.	Discont.	–	–	–	Old ant.-sept.	1	
9	B. M.	M	58	2	1 yr.	Discont.	–	–	–	Normal	None definite	
10	J. K.	M	41	2	5 yr.	Restrict.	–	–	–	Old post.	1	
BECK I PLUS VINEBERG												
11	E. C.	F	55	½	3 yr.	Discont.	–	–	–	Old post.	1	
12	J. L.	M	44	3	1 yr.	Discont.	–	–	–	Old post.	History vague	
13	D. C.	M	54	½	3 yr.	Discont.	Exertion	+++	General-ized	L. vent. hypertrophy; old ant.-sept.	3	

(*For footnote, see page 74.*)

CASES OF CORONARY ARTERY DISEASE TREATED BY SURGICAL REVASCULARIZATION

	POSTOPERATIVE							
OPERATIVE AND POSTOPERATIVE COMPLICATIONS	ANGINA*	EMPLOYMENT	DYSPNEA	CONGESTIVE FAILURE	CARDIAC ENLARGEMENT	LONG-TERM ECG	RESULTS	REMARKS
	None	Full	None	–	–	No ess. change	Excell.	
Pulmonary embolus after second stage	None	Full	–	–	None	No ess. change	Excell.	
	None	Full	–	–	–	No ess. change	Excell.	Severe congestive failure after 2nd stage; 3rd operation revealed large aorticosinus fistula; occlusion of sinus gave excellent result
Patient died of cardiac arrest immediately after completion of first stage								
Died; "blowout" of vascular graft on the 13th PO day after 1st stage; received anticoagulants postoperatively								
Postoperative empyema	Minimal	Full	None	None	None	No ess. change	Excell.	
Pulmonary infarction	None	Full	None	–	–	No ess. change	Excell.	
Hiccough, belching, abdominal pain and distention; thrombophlebitis	None	Full	None	–	–	No ess. change	Excell.	Abdominal and lower intercostal chest pain but no angina; symptoms of diaphragmatic irritability persist
Hiccough, abdominal pain, gastric distress	5 blocks	Restrict.	–	–	–	No ess. change	Fair	
Hiccough, gastric distress, abdominal distention	5 blocks	Restrict.	–	–	–	No ess. change	Fair	Symptoms of diaphragmatic irritability
	None	Full	–	–	–	No ess. change	Excell.	
Thoracentesis; 350 c.c. serous fluid	None	Full	–	–	–	No ess. change	Excell.	
Died of cardiac arrest on operating table as internal mammary artery was being implanted into myocardium								Because of advanced myocardial fibrosis and advanced arteriosclerosis, the patient would not now be considered a candidate for surgery

TABLE I—CONTINUED

CASE NO.	NAME	SEX	AGE	PREOPERATIVE							
				ANGINA*		EMPLOYMENT	DYSPNEA	CONGESTIVE FAILURE	CARDIAC ENLARGEMENT	ECG	NO. OF PREVIOUS MYOCARDIAL INFARCTIONS
				THRESHOLD (BLOCKS)	DURATION						
VINEBERG											
14	S. F.	M	55	½	7 yr.	Discont.	Exertion	–	–	Old ant.	1
BECK I											
15	A. H.	M	32	2	1½ yr.	Discont.	–	–	–	Old post.	2
16	F. S.	M	64	4	15 mo.	Discont.	–	–	–	Old post.	2
17	G. J.	F	53	3	15 mo.	Discont.	–	–	–	Normal	2
18	J. T.	M	39	½	3 yr.	Restrict.	Exertion	–	L. vent.	Old ant.	1
19	W. C.	M	41	2	6 mo.	Discont.	–	–	–	Old ant.-sept.	1
20	V. S.	M	57	½	6 mo.	Discont.	Exertion	–	–	Old post.	1
21	J. C.	M	45	5	3 yr.	Restrict.	–	–	–	Normal	None
22†	W. C.	M	44	2	2 yr.	Discont.	Exertion	–	L. vent.	Old ant.-sept.	2
23†	S. S.	M	50	½	2 yr.	Discont.	Exertion	–	L. vent.	Right bundle branch block; old ant.	1
24	D. M.	M	44	2	1 yr.	Restrict.	Exertion	+	L. vent.	Normal	1
25	R. A.	M	34	2	1 yr.	Restrict.	–	–	–	Old post.	2
26	T. E.	M	41	1½	3 yr.	Restrict.	–	–	–	Old post.	1
27†	A. A.	M	38	2	3 yr.	Restrict.	–	–	–	Old post.	History vague
28†	N. R.	M	40	½	2 yr.	Restrict.	Exertion	–	–	Old ant.	2
29†	J. K.	M	43	5	1 yr.	Restrict.	–	–	–	Old post.	1
30†	H. G.	M	48	1	8 mo.	Discont.	–	–	–	Old post.	1
31†	M. C.	F	53	3	2 yr.	Restrict.	–	–	–	Old ant.-sept.	2
32†	J. B.	M	54	½	3 yr.	Restrict.	–	–	–	Old ant.-sept.	2

*Measured as ability to walk city blocks.

†Cases 22, 23, 27-32 had first stage Beck II operation. Thrombosis of the vascular graft was noted at the time of the second stage operation. A Beck I procedure was performed at the time of the second operation.

TABLE I—CONT'D

	POSTOPERATIVE							
OPERATIVE AND POSTOPERATIVE COMPLICATIONS	ANGINA*	EMPLOYMENT	DYSPNEA	CONGESTIVE FAILURE	CARDIAC ENLARGEMENT	LONG-TERM ECG	RESULTS	REMARKS
	½ block	Discont.	None	–	–	No ess. change	Poor	Died 18 mo. after operation
Operative and postoperative sinus tachycardia	None	Full	–	–	–	Improved T waves	Excell.	
Acute anteroseptal infarction on the 5th PO day	None	Full	–	–	–	No ess. change	Excell.	
Cardiac arrest on the operating table; hypotension until 5th PO day; severe angina on 19th PO day	None	Full	–	–	–	No ess. change	Excell.	
Sinus tachycardia for 13 days postoperatively	None	Full	None	–	None	No ess. change	Excell.	
	None	Full	–	–	–	No ess. change	Excell.	
Ventricular tachycardia during anesthesia; surgery 2 mo. later; developed ventricular tachycardia during abrasion of epicardium	None	Full	None	–	–	No ess. change	Excell.	
Bronchopneumonia; mild	None	Full	–	–	–	No ess. change	Excell.	
Hemothorax on 8th PO day after first stage Beck II operation	5 blocks	Restrict.	None	–	None	No ess. change	Good	
	None	Full	None	–	None	No ess. change	Excell.	
	None	Full	None	–	None	No ess. change	Excell.	
	None	Full	–	–	–	No ess. change	Excell.	
	None	Full	–	–	–	No ess. change	Excell.	
	2 blocks	Restrict.	–	–	–	Repeated myocardial infarcts	Poor	Relief of symptoms for 18 mo.; angina then returned; has had 3 coronary occlusions since surgery
	None	Full	None	–	–	No ess. change	Excell.	
	None	Full	–	–	–	No ess. change	Excell.	
	Minimal	Full	–	–	–	No ess. change	Excell.	
Postoperative atelectasis	None	Full	–	–	–	No ess. change	Excell.	
	None	Full	–	–	–	No ess. change	Excell.	

Hypertension.—Patients with more than a mild or moderate degree of hypertension were not considered for surgery. Only one patient (Case No. 19) had a past history of hypertension, and he was normotensive at the time of surgery.

Operative Complication.—The most common operative complication was the appearance of cardiac arrhythmias during the induction of anesthesia and during surgery. Although many types of arrhythmia were observed the most frequently encountered were nodal extrasystoles, nodal rhythm, interference dissociation, and auricular and ventricular premature contractions. These abnormalities appearing early in the operation were not considered to be hazardous and usually subsided with either adequate oxygenation or temporary cessation of the operative procedure. Ventricular extrasystoles uniformly appeared during abrasion of the epicardium and during procedures involving manipulation of the ventricles.

There were four instances of serious cardiac arrthyhmia. Cardiac arrest occurred in 3 patients. Two of these died; one on the operating table and the other on returning to his room. The third patient is alive and free of symptoms. In one case ventricular tachycardia occurred during the induction of anesthesia. Surgery was canceled and the tachycardia reverted spontaneously. When surgery was performed two months later, ventricular tachycardia appeared during abrasion of the epicardium. Intravenous procaine amide controlled the rhythm and the patient had an uneventful postoperative course.

Operative Mortality.—There were three operative deaths among the 32 patients, giving an operative mortality of 9.4 per cent. Two of the deaths were in the younger age group and were among the first 6 patients subjected to surgery. One patient died of cardiac arrest at the conclusion of the first stage of the Beck II operation. The second patient had a "blowout" of the aorticosinus graft on the thirteenth postoperative day. This was the only patient who received anticoagulants postoperatively. The third patient died of cardiac arrest during a combined Beck I and Vineberg operation. Left bundle branch block preceded the appearance of cardiac standstill and appeared as the internal mammary artery was being implanted in the ventricular myocardium. In the light of our present concepts, this patient would not now be considered a candidate for surgery since he had evidence of generalized arteriosclerosis, congestive failure, and marked cardiac enlargement.

There were no deaths among those patients undergoing the Beck I operation.

Postoperative Complications.—In all cases special care and attention had to be directed to the respiratory system. Retention of bronchial secretions with subsequent pneumonitis and localized areas of atelectasis were frequent, but not alarming, postoperative complications. In one case, bronchopneumonia was of sufficient magnitude to retard a smooth postoperative course. Significant atelectasis developed in one patient, and empyema occurred in one. Although small quantities of pleural fluid developed in almost all cases,

a thoracentesis was required on only two occasions. In one of these hemothorax was present. Postoperative pulmonary infarction occurred in 2 cases. Gastric symptoms and signs of diaphragmatic irritability were noted after omentopexy.

One patient had postoperative hypotension of such a degree that Levophed had to be administered by continuous intravenous drip until the fifth postoperative day. Two patients developed sinus tachycardia. In one, it was mild and transitory. In the other, it persisted until the thirteenth postoperative day. In neither case was there any serious consequence of the tachycardia.

All patients but 2 were relieved of their angina during the immediate postoperative period. One patient, who had postoperative angina, developed an acute anterior myocardial infarction on the fifth postoperative day. He recovered and has returned to full-time work without disability. The other patient had severe angina on the nineteenth postoperative day.

Signs of pericarditis, as indicated by the appearance of either a friction rub, chest pain, or electrocardiographic changes were observed in all cases. The friction rub was audible almost immediately after surgery and persisted until the fourth or fifth postoperative day. In no case was there significant pericardial effusion or evidence of constrictive pericarditis.

In 8 (Cases 22, 23, 27-32) of the 15 patients who underwent the Beck II operation, thrombosis of the vascular graft was found at the time of the second-stage operation. A Beck I procedure then was performed in these cases. Two patients who submitted to the first stage of the Beck I operation refused the second stage. In only 3 cases was the graft known to be patent.

Electrocardiogram.—An abnormal electrocardiogram, including definite evidence of previous myocardial infarction, was obtained in 28 of the 32 cases. Two patients had right bundle branch block. There were no cases of left bundle branch block. All patients had a regular sinus rhythm. Electrocardiograms taken on the second postoperative day showed either no change in comparison with preoperative tracings or evidence of pericarditis. The changes attributed to pericarditis usually disappeared by the end of the first month. In general, late postoperative graphs were essentially unchanged over preoperative tracings.

Long-Term Deaths.—Of the 29 patients who survived surgery and who have been followed up to five years, only one has died since surgery. This patient had severe coronary artery disease with persistent angina. He died eighteen months after a Vineberg operation.

DISCUSSION

Two types of death result from coronary artery disease. The first is due to the appearance of ventricular arrhythmias. The second is the result of progressive myocardial degeneration. The former accounts for 90 per cent of deaths due to coronary artery disease. In many of these cases the actual pathologic damage to the heart is usually slight, and it would appear that

the heart could still function adequately if only the rhythm could be protected. Arrhythmias appear when areas of unequal oxygenation of the myocardium are adjacent to one another. Harris and Rojas[5] demonstrated that ventricular extrasystoles arise from this area. Brofman and his associates[6] have been able to demonstrate that unequal oxygenation of the heart can result in ventricular fibrillation and death. Protection is dependent, in part, upon an equitable distribution of oxygenated blood throughout the myocardium. The intercoronary channels are an important mechanism of protection. They may develop naturally during the course of coronary artery disease. Failure of these anastomoses to develop can result in sudden death or progressive myocardial degeneration. One of the major objectives of surgical revascularization of the heart is to promote increased utilization of existing intercoronary channels and to stimulate the development of new anastomotic communications that will provide a more equitable distribution of oxygenated blood, and thereby protect the heart from the serious effects of coronary artery disease.

It must be realized that revascularization procedures do not stop or revert the progress of the vascular arteriosclerotic process. Dead muscle cannot be revitalized or replaced. Consequently, surgical revascularization cannot be applied to all patients having coronary artery disease. Those patients in whom the disease process has progressed to such a degree as to cause irreversible myocardial degeneration, or those patients in whom revascularization of the heart cannot keep pace with a rapidly advancing disease process, will not benefit from surgery. Those patients who present evidence of moderately advanced coronary artery disease as manifested by a slight degree of cardiac enlargement and minimal congestive failure in the presence of stabilized angina of effort represent borderline cases. As was observed in this series, the results are usually beneficial but a good result cannot always be expected.

The operative mortality for surgical revascularization of the heart is not unduly high, considering the nature of the disease process. The over-all operative mortality in this series was 9.4 per cent. Two of the deaths occurred in the early years of the study. There was only one death among the last 26 cases, resulting in a mortality rate of 3.8 per cent. A careful and intelligent choice of candidates for surgery and the selection of the proper surgical procedure for the individual case will lower the operative mortality and increase the number of patients benefited by revascularization of the heart. In spite of these precautions, and because of the seriousness of the disease process, it is unlikely that the operative mortality will be reduced below a certain minimal level.

Postoperative complications were not excessive. Careful attention to the physiology of the respiratory system can alleviate greatly the frequency and severity of postoperative complications. The most common problems are those related to retained bronchial secretions with subsequent atelectasis and

pneumonitis. Although surgery is performed on a diseased heart, the complications arising from the cardiovascular system are less than would be anticipated.

The results of surgery in this series of patients have been most gratifying. In over five years of postoperative observation, there has been only one long-term death. Of the surviving patients, 82.7 per cent have been almost completely relieved of angina and have been able to return to their former occupations. Two of the patients in this series have shown little improvement, but are in no worse condition than preoperatively. Only 2 of the patients in the series had a poor result.

Greater emphasis should be placed on the principles of coronary artery surgery rather than upon individual surgical techniques. Any operation that is designed to revascularize the heart should be tested in the animal laboratory before being applied to the human being. The Beck I operation fulfills the laboratory criteria for a successful surgical procedure. Clinical experience indicates that this operation permits a rather wide latitude in application. The operative mortality is low and the results of surgery have been favorable. Combined procedures are not recommended for routine use.

SUMMARY AND CONCLUSIONS

1. The results of the surgical treatment of coronary artery disease in 32 cases together with a five-year follow-up are presented.

2. Surgical revascularization of the heart in properly selected patients is an important and valuable method of treatment of coronary artery disease.

3. Laboratory experiments and clinical experience indicate that the Beck I operation, at present, is the procedure of choice in the surgical treatment of coronary artery disease.

The authors wish to extend their appreciation to the New Jersey State Department of Health for the cooperation and assistance received in the performance of this study.

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CASE OF GRANULAR-CELL MYOBLASTOMA (ORGANOID TYPE) INVOLVING ARM, LUNG, AND BRAIN, WITH TWENTY YEARS' SURVIVAL

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SURVIVAL of twenty years or more is rare in a case of malignant tumor, proved histologically, after extensive and multiple metastases.

INTRODUCTION

In 1953, a long survival with a *malignant melanoma* was reported by Galgano.[9] It was unusual because of the long time between the detection of the primary lesion and the subsequent first appearance of pulmonary metastases, and the unusually long time between the development of metastases in the lung and the death of the patient. The primary lesion was in the skin of the right temple and proved to be a melanoma histologically. It was not until eleven years later that any pulmonary symptoms developed showing, by x-ray examination, a small nodule in the left lung. There was an additional long interval of nine years between the detection of pulmonary metastases by x-ray examination and the death of the patient, in all, a period of forty years from the development of the original skin lesion until a fatal issue ensued. The patient finally died at the age of 86 (her first skin lesion having developed at the age of 46) of widespread pulmonary and other metastases.

In 1952, Ficarra[7] described a similar case (with post-mortem examination) of a patient, twenty years after a bilateral mastectomy for carcinoma. The author presented his case as an instance of so-called "tissue resistance" to cancer. There must be, he thought, some type of immunity or pseudo-immunity to cancer in certain fortunate individuals to account for the long course of disease occasionally seen in verified cases of malignancy. No matter what term is used, many clinicians have observed that of 2 patients with the same type of cancer, of approximately the same age, with the same duration of the primary disease, one patient may die within a year or less and the other may survive for decades. In the case reported by Ficarra, that of a 60-year-old woman, the patient had had a bilateral mastectomy for carcinoma twenty years previously and had not received any x-ray therapy postoperatively. She had carried out her usual family duties, her health had been good for almost twenty years after the bilateral mastectomy until several months before her death when she developed loss of weight and a nonproductive cough. One month before her

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Read at a regular meeting of the Richmond (Va.) Academy of Medicine, March 13, 1956.
Received for publication Jan. 19, 1957.

hospitalization, ascites developed. At autopsy, cancer nodules were found from head to knees and at both elbows. No metastases were found in the extremities beyond the elbows or knees. The skeleton and the viscera were studded with adenocarcinoma. One of the photographs shows the omentum which was literally riddled with metastatic cancer nodules. The author explains the absence of metastases below the elbows and knees by the investigations of Batson who, in 1940, showed the function of the vertebral veins and their importance in the spread of metastases. These veins extend from the coccyx to the skull and communicate with the veins in the ribs, shoulder girdle, pelvic girdle, upper end of the humerus and femur. In this case, the lesion was the usual variety of adenocarcinoma of the breast. It was not even known, the operations having been carried out in a distant country, whether *each* breast was involved originally in carcinoma.

Hutcheson[11] reported, in 1952, a case of metastatic cyst-adenocarcinoma of the ovary thirty-three years after removal of the primary growth. This is also an extreme example of the ability of malignant cells to remain dormant for long periods and then to proliferate again. This particular tumor caused death thirty-three years after removal of the primary pelvic growth without having given rise to symptoms during the long interim. Recurrence, verified at postmortem examination, in the pelvis and lower abdomen was detected only six months before death. Edema of the lower extremities then developed. The patient had been entirely well from the time of the original operation and tumor removal thirty-three years previously until the terminal edema. A complete necropsy showed no other primary tumor.

Reyes and Horrax[15] described (1950) a case of metastatic (brain) melanoma extirpated from the left frontal lobe, which was the size of a duck's egg, after previous melanomas had been removed from the breast and axilla in a 57-year-old man. The patient was alive, well, and working as an insurance salesman almost four years later. The *original* dark mole had been widely excised from the left shoulder two years before craniotomy.

These cases are examples of rather common types of malignancy, but the case to be reported, as the title of this paper suggests, is of *obscure histologic origin* and therefore of added interest for that reason alone.

CASE REPORT

C. H., a 35-year-old white man (referred by Dr. Arthur Brinkley, of Richmond, Va.), was admitted Aug. 9, 1952, for the first time to the Medical College of Virginia Hospital for treatment of presumably metastatic tumors of the left lung. Past history disclosed that in 1935, when the patient was 18, a mass was removed from the right biceps muscle in another hospital. After considerable microscopic study, the final diagnosis at that time was hemangio-endothelioma. In the spring of 1936, x-ray films of the chest revealed three nodules in the left lung and several questionable nodules in the right lung. The patient was given 500 r of x-ray therapy to the lungs from the spring of 1936 to the spring of 1937. Subsequent periodic chest x-ray examinations had continued to show a persistent shadow in the basilar region of the left lower lobe, and several rather questionable densities in the right lung field. There had been a slow, progressive enlargement in the left lower lobe lesion from 2 cm. in 1937 to 4 cm. in 1952 (Figs. 1 and 2).

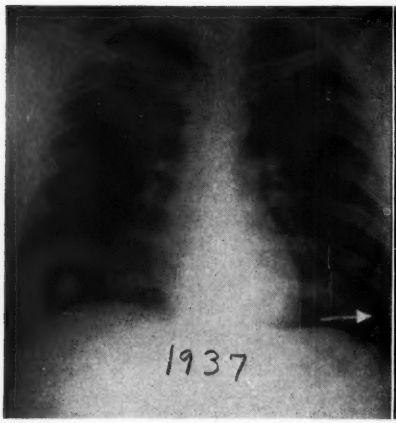

Fig. 1.

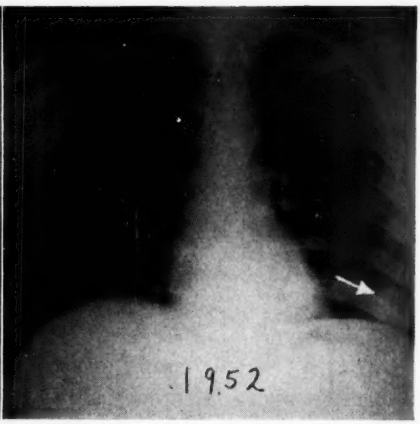

Fig. 2.

Fig. 1.—Roentgenogram of chest made in 1937 which shows well-defined round density in left lower lobe (arrow).

Fig. 2.—Roentgenogram of chest made in 1952 which shows increased size of density noted in 1937 film (arrow).

Fig. 3.—Ventriculogram showing distortion and compression of right lateral ventricle by intracerebral metastatic tumor, with shift of ventricular system to the left.

The patient had remained asymptomatic during these years and had had no cough, weight loss, chest pain, or hemoptysis. There had been no evidence of local recurrence of the primary lesion. During the past two months, however, the patient had complained of daily frontal headaches and bilateral tinnitus, neither of which he had ever had before. He was seen by the neurosurgeons prior to his present admission and no objective evidence of intracranial metastasis or of increased intracranial pressure was found.

Physical examination revealed a temperature of 98.2° F., pulse 72, and respirations 16. The lungs were clear to percussion and auscultation with no râles present. Results of all other examinations were also negative.

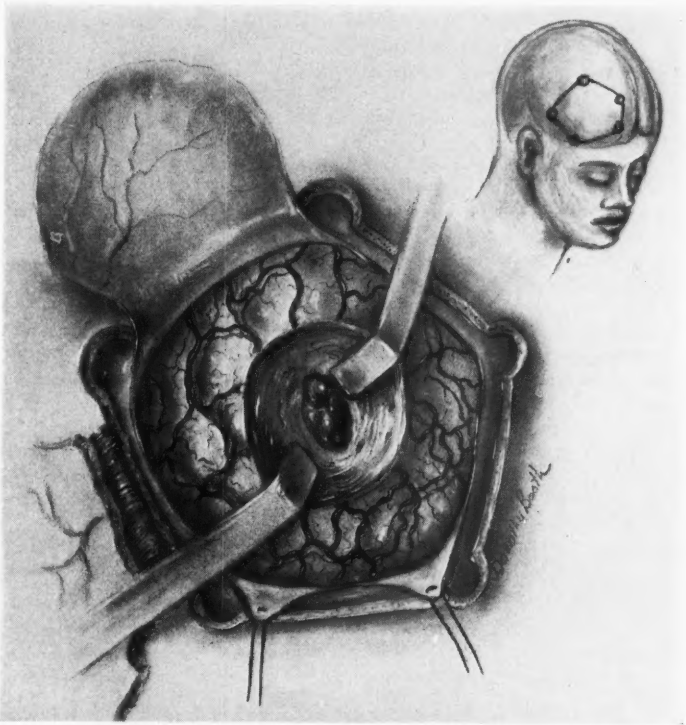

Fig. 4.—Artist's drawing showing location and appearance of single deep metastatic lesion in posterior aspect of right frontal lobe. The smaller figure shows the location of the bone flap.

Laboratory findings were noncontributory, except for the chest roentgenogram. This revealed a well-rounded nodule in the region of the lateral basal segment of the left lower lobe. No definite nodules were seen in the right lung fields at this time.

On the third hospital day, a left thoracotomy was done. A large nodule measuring approximately 4 cm. in diameter was found in the lateral basal segment of the *left* lower lobe. There were two smaller nodules, each less than 1 cm. in diameter, in the anterior and posterior segments of the left upper lobe, respectively. A basal segmental resection and wedge resection of the smaller nodules in the upper lobe were done.

The patient was discharged Aug. 23, 1952, but two days prior to discharge, a planogram of the *right chest* revealed an area of density 2 cm. from the posterior wall which

suggested a metastatic nodule. The patient was to return in three weeks for an exploratory thoracotomy on the right side.

However, during the interval at home, the headaches which the patient had experienced prior to chest surgery returned (one week following discharge). These were more severe than previously and were confined to the frontal and occipital regions. There were no localizing neurological symptoms. The patient was hospitalized again at the Medical College of Virginia Hospital on Oct. 2, 1952, for further study.

Physical examination then revealed a bilateral papilledema of three diopters. The reflexes were slightly more active on the left side. A craniotomy was performed on Oct. 9, 1952, for a "probable metastatic lesion in the right frontal lobe," the ventriculogram having suggested such a lesion (Fig. 3) earlier on the same day.

After opening the dura, a soft area was evident in the right posterior frontal region. A circular wedge-shaped portion of the cerebral cortex and subjacent white matter was removed down to and overlying a firm, well-circumscribed, small tumor nodule, about 2 cm. in diameter (Fig. 4). The mass was enucleated in its entirety. The bone flap was discarded because of cerebral edema. Portions of the tumor were sent in a sterile container to the tissue culture laboratory at the Columbia-Presbyterian Medical Center, New York City (see below). The remainder was utilized for pathologic examination.

The postoperative course was entirely satisfactory and the patient was discharged twelve days after the operation, to be followed as an outpatient. A thoracotomy on the right side was still contemplated.

After a tantalum plate insertion in the skull (December, 1952), the patient having remained symptomatically free, the right lung was explored on Jan. 22, 1953. The entire right lung was carefully palpated and a very small nodule (2 to 3 mm. in diameter) was found and removed from the inferior margin of the middle lobe. This was microscopically identical with the left pulmonary lesion removed in August, 1952.

In 1954, he developed definite signs of increased intracranial pressure and it was obvious that he had multiple intracranial metastases for a year or more before death. The patient was then given x-ray treatment to the brain: April to August, 1954 (3,000 r—tumor dose). A high degree of choked discs developed bilaterally and he became blind several weeks before he died at his home on June 3, 1955. There were several external discrete nontender but firm swellings on the scalp at the time of death. A post-mortem examination unfortunately was not obtainable.

Pathologic Findings.—

We were fortunate in having available a slide of the lesion removed from the forearm in 1935. This showed alveolar groupings of tumor cells, the groups being separated only by thin-walled, vascular spaces (Figs. 5 and 6). The cells of the groups were large, but fairly uniform with large, bizarre nuclei and prominent nucleoli. A pseudoglandular pattern was sometimes distinguished, but this was thought to be artifactual.

1. *Lung lesions, August, 1952:*

Gross (S-52 5371): The specimen consisted of the basal segment of a lung 11 by 13 by 9 cm. and weighing 99 Gm. The pleura was smooth, and a mass was palpable in the region of the diaphragmatic pleura. On cut section, a well-circumscribed tumor 3 by 2.7 cm. was found (Fig. 7). This had a gray-pink color, a fibrous texture, and showed focal hemorrhage. The bronchus leading toward the tumor was normal. In addition, there were three different portions of tissue. The first was from the upper lobe, measured 2.5 by 2 cm. and showed, on cut surface, a sharply circumscribed nodule 6 by 2 mm. The second specimen, also from the upper lobe, measured 2.5 by 0.3 cm., and contained a similar nodule 2 mm. in diameter. The final tissue was a peribronchial lymph node 1.8 by 1 cm. showing no unusual features.

Microscopic: Sections of the tumor mass and smaller nodules showed similar features. They were obviously metastatic lesions and almost identical with that noted in the lesion removed in 1935 (Figs. 8 and 9). Innumerable pseudo-alveoli rather closely

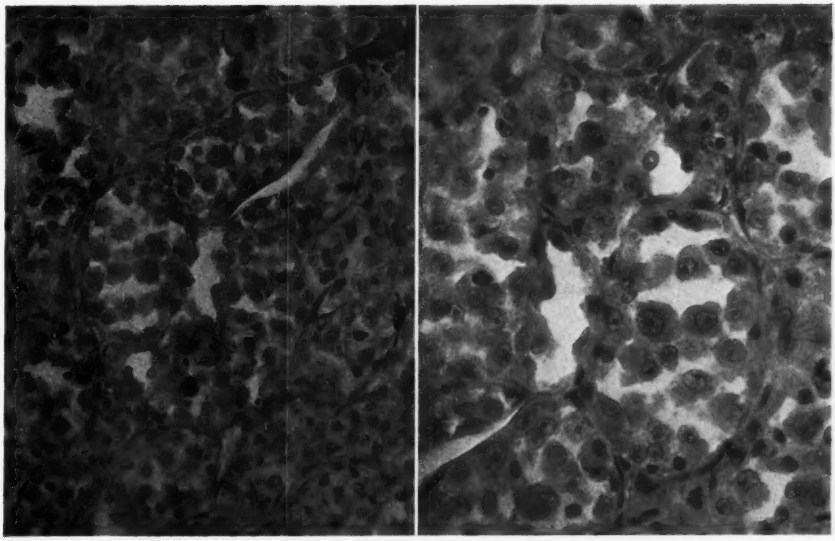

Fig. 5. Fig. 6.

Fig. 5.—Tumor from forearm to show pseudo-alveolar grouping of tumor cells in a highly organoid pattern.

Fig. 6.—High-power view of Fig. 5 to show details of the individual tumor cells. Coarse and fine granules are present in the cytoplasm.

Fig. 7.—Basal segment of left lower lobe of lung with well-defined nodule of metastatic tumor beneath the diaphragmatic pleura.

Fig. 8.

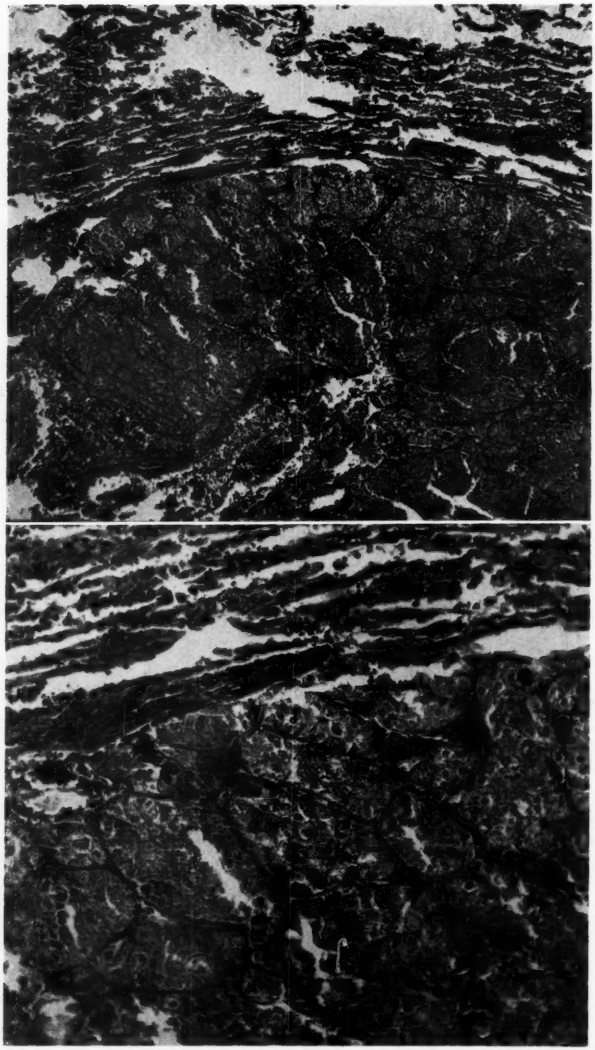

Fig. 9.

Fig. 8.—Photomicrograph of metastatic lung tumor to show similar organoid pattern to that of the primary growth.

Fig. 9.—Medium-power view of Fig. 8. Compare with Fig. 5.

placed were seen; these were made up of sheets of large, granular cells with rounded vesicular nuclei. The granularity of the cells varied, being fine and evenly distributed in some cells, while in others in was condensed into small, amorphous aggregates. Stains for mucin were negative, but a fat stain showed appreciable droplets of fat in some areas.

2. *Cerebral lesion, October, 1952*:

Gross (S-52 6667): The specimen consisted of a small, encapsulated, nearly spherical, firm, grayish-pink tumor, to which was attached bits of blood-stained brain tissue (Fig. 10). The tumor measured 1.8 by 1.5 by 1 cm. and weighed 3 Gm. The cut surface was homogeneous, magenta, firm and smooth, and surrounded by a capsule, 0.5 to 1 mm. in thickness.

Fig. 10.—Hemisection of metastatic tumor from frontal lobe.

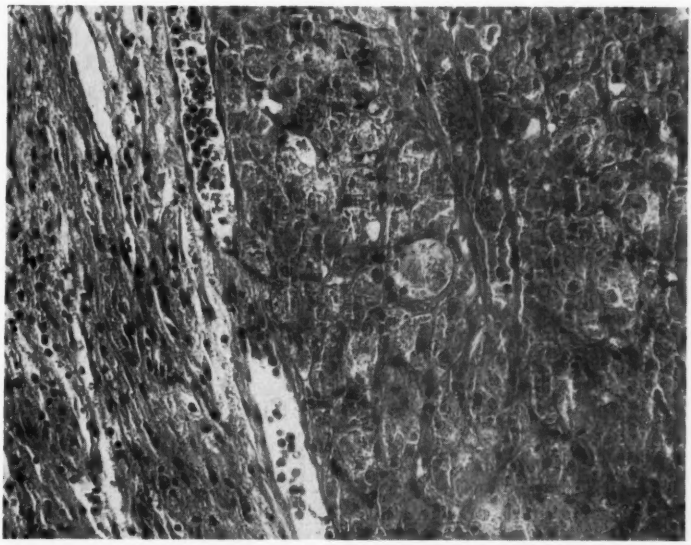

Fig. 11.—Metastatic brain tumor to show compression of brain tissue on left. Note granules in cytoplasm of tumor cells.

Microscopic: The features were similar to that noted above, and need no further elaboration. Despite the gross impression, encapsulation was rather poor (Fig. 11).

3. *Lung lesions, January, 1953:*

Gross (S-53 521): The specimen consisted of two small pieces of bluish-colored lung tissue from the right middle lobe, and a small white piece of tumor tissue 5 by 4 by 3 mm.

Microscopic: The same organoid pattern as noted in the previous surgical specimens was maintained.

Final Diagnosis.—Granular-cell myoblastoma, organoid type, of biceps muscle with metastasis to both lungs and right frontal lobe of brain.

DISCUSSION

This tumor is an interesting one about which there is some difference of opinion among pathologists as to its histogenesis. Three names have been used to describe it, namely, alveolar soft part sarcoma, malignant granular-cell myoblastoma of the organoid type, and malignant nonchromaffin paraganglioma.

Alveolar soft part sarcoma was favored by Christopherson, Foote, and Stewart,[3] and these authors reported 12 cases in 1952. Six of the patients developed metastases and, of these, 4 had died at the time of their report. The authors favored the term "alveolar soft part sarcoma" because it was descriptive in the histologic sense and noncommittal concerning its histogenesis.

Smetana and Scott[17] believed that the tumor, of which they studied 14 cases, was derived from nonchromaffin paraganglia. To support their thesis they cite the close relationship of these growths with nerves and their histologic similarity to carotid body tumors. In addition, the authors make reference to the finding by Lent Johnson of vascular glomera structurally similar to the carotid body in relation to the blood vessels of Hunter's canal.

Stout[18] took issue with the term of nonchromaffin paraganglioma. He did not entirely reject Smetana's hypothesis but still felt unconvinced since paragangliomas of the carotid body type almost never metastasize, while the tumor under discussion is regularly malignant. Stout's experience with the organoid variety of tumor comprised 11 cases studied in the laboratory of Surgical Pathology at Columbia University.

Willis[19] emphasized that he was uncertain as to the true nature of this tumor. He thought, in fact, that the term "myoblastoma" was being used as a "dumping ground" for all sorts of large granular-cell tumors, probably of *diverse* origins.

This paper does not propose to resolve the argument concerning the nature of this tumor. However, as mentioned previously, the cerebral metastatic material was submitted to Dr. Margaret Murray's tissue culture laboratory at Columbia University with the following final report from Dr. Murray:

> This tumor grew quickly and well, and throughout the 51 days that it was kept in vitro showed frequent, usually normal, mitoses. The tendency to proliferate may have been working against cell differentiation. The cells were of a general mesoblastic appearance and varied considerably in size. There was from time to time a bewildering variety of cell forms; but the most frequent, recurrent and recognizable specialization was in the direction of skeletal muscle: long, ribbon-shaped cells with abundant longitudinal striae were characteristic. These forms developed in vitro from the small, roundish,

relatively compact cells which made up the explanted tumor nodules. Their nuclei also developed in vitro from being small and round, with 1 or 2 nucleoli, to being larger, oblong and vesicular, with several nucleoli. These changes are reminiscent of regenerating skeletal muscle, and of the behavior of adult skeletal muscle in vitro. Multinucleate cells, anastomosing ribbons, and large cells containing bizarre multilobate nuclei are also present. These too are consistent with the picture of skeletal muscle—both normal and neoplastic.

This could be a *granular cell myoblastoma.* Apparently these tumors are rather protean in their manifestations in vitro—but most of the characteristic cell types seen in cultures of them are also characteristic of some form of skeletal muscle. This principle applies in this tumor. From its behavior in vitro, I do not see any grounds for aligning this tissue with the paraganglioma.[14]

Kelly and Langston[12] totaled the known cases of pulmonary resection for metastatic malignancy and found 109 instances with a two-year survival rate of 23.2 per cent. A number of long-term "cures" were reported, however. The results with sarcomas have been at least as satisfactory as with metastatic carcinoma.

Bilateral resection for metastatic pulmonary malignancy has been performed in only a few cases. Alexander and Haight's patient[1] who had bilateral pulmonary lobectomy after removal of a neurogenic sarcoma of the forearm was known to be well twelve years after the second lobectomy. Hood, McBurney, and Clagett[10] recorded a patient who had bilateral pulmonary resection for metastases eleven and fourteen years after excision of a fibrosarcoma of the leg. She was well forty months after the last operation. Churchill[4] mentioned a patient who had been treated with bilateral partial lobectomy. In addition to the patient reported here, we are familiar with 3 additional unreported cases of bilateral pulmonary resection for metastatic malignancy.[2, 5, 8]

Efforts to eradicate both pulmonary and cerebral malignant deposits are not likely to prove successful except in the very rare instance. This aggressive surgical approach could be expected to yield best results in the patient whose primary lesion is located in the lung. In the London Hospital autopsy records there were 85 cases of primary carcinoma of the lung associated with cerebral metastases. In 26 of the 85 cases, a solitary cerebral metastasis was demonstrated. Sporadic reports have appeared in the literature describing excision of apparently *solitary cerebral metastases* some time after pulmonary resection for primary lung tumor.

Reports of excision of both metastatic pulmonary and cerebral lesions are far more rare. Effler and Blades[6] reported a case of adenocarcinoma of the colon with resection of the primary lesion, subsequent pulmonary lobectomy for metastasis and finally excision of a cerebral metastasis two years after removal of the primary tumor.

A remarkable case was described by Moore, Perese, and Staubitz[13] in which removal of a testicle for teratocarcinoma was followed successively by craniotomy, pneumonectomy, two additional craniotomies and, finally, by excision of an isolated jejunal lesion. The patient was alive and well at the time of the report four years after the orchiectomy.

The accumulated experience in the treatment of multiple metastatic pulmonary malignancy is obviously rather meager. We are unaware of any previous attempt at bilateral pulmonary resection with excision of cerebral metastasis as well, such as was performed on our patient.[16]

SUMMARY

In summary, the case of a white man is presented who, at the age of 18, developed a soft tissue tumor in the right biceps muscle. This was excised and was followed 17 years later by surgically verified bilateral pulmonary and cerebral metastases with fatal issue almost three years after the removal of the latter lesions and twenty years after the original appearance and excision of the soft tissue muscle mass. In our opinion, the tumor was a granular-cell myoblastoma of the organoid type. This interpretation was in part supported by the results of tissue culture studies of the cerebral metastasis.

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SPONTANEOUS BILATERAL CHYLOPNEUMOTHORAX

A Case Report

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SPONTANEOUS chylopneumothorax is a rare condition. We have been unable to find any reported case of spontaneous bilateral chylopneumothorax in the American or British literature and have found only one case of spontaneous chylopneumothorax which was unilateral.[1] This case, which followed lifting, was treated conservatively with recovery. It was postulated that the patient had ruptured a pulmonary bleb, developed a spontaneous pneumothorax, and had torn a lymphatic collateral, giving rise to the chylous fluid.

The following case of bilateral chylopneumothorax was found to be due to a diffuse pleural mesothelioma. Pleural mesothelioma is of infrequent occurrence and no report of a case with chylous fluid was found in a review of the American literature.

CASE REPORT

C. C. N., a 25-year-old white man, was admitted to Birmingham Veterans Administration Hospital on April 28, 1956, with a chief complaint of shortness of breath for one week and dyspnea for twenty-four hours. He stated that he had lost weight and strength for a year. Exertional shortness of breath had been present for four to six months. There was no pleural pain, no history of trauma, and the past history was negative.

The review of systems was not significant.

The patient's father had died of carcinoma of the stomach, and all of his father's family had died of cancer. There was no family history of tuberculosis.

Physical examination revealed a small, thin, white man in acute respiratory distress. Respirations were labored and there was slight intercostal retraction on inspiration on the right. Examination of the head, eyes, ears, nose, and throat revealed no abnormalities. There was no neck vein distention and no cervical lymph nodes were palpable. The trachea deviated slightly to the right. The entire right chest was dull to percussion and the left base also. Tactile fremitus was reduced and breath and voice sounds absent over these same areas. No friction rub was heard. Clubbing of the fingers, with cyanosis of the nail beds, was noted. The remainder of the physical examination was negative. Fluoroscopy revealed a fluid level up to the fourth intercostal space on the right. The initial impression of the ward physician was pleural effusion due to tuberculosis or lymphoma. Thoracentesis on the right yielded 1,400 c.c. of milky white fluid. The dyspnea decreased. A continuous air leak was present so a large gauge needle was left in the pleural space and attached to water-seal drainage. With suction, no re-expansion of the lung occurred. A surgical consultation was obtained and review of the initial chest x-ray revealed the presence of air and fluid in both pleural spaces with partial collapse of both lungs. Intercostal catheter suction on the right was instituted but did not re-expand the lung. The chylous fluid continued to drain in large amounts. Sudan III stain of the fluid indicated a high fat content. Cell studies on

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Received for publication Feb. 1, 1957.

the fluid revealed large numbers of mononuclear white cells and individual mesothelial-type cells with no malignant characteristics. The patient continued to lose 2,000 to 3,000 c.c. of chyle daily. He maintained a good oral intake of fluid and solid foods.

More air began to accumulate in the left pleural space and dyspnea increased. Intercostal catheter drainage on the left removed 2 L. of chyle and much air. Ventilation improved. With clearing of the right pleural space of all fluid, enlargement of the right superior mediastinal shadow was seen on x-ray. Bronchoscopy was negative. Thoracotomy was performed to arrive at a definite diagnosis, to attempt to close the air leaks, decorticate and re-expand the right lung and, also, to control the debilitating chylothorax. A preoperative diagnosis of bilateral chylothorax of unknown etiology, possible mesothelial cell tumor, was made.

At operation on the right, all pleural surfaces were extensively replaced by tumor growing in sheets with some areas thickened up to form trabeculations. The lung was about 75 per cent collapsed, affecting all three lobes equally. The visceral pleura was replaced by a thin layer of this tissue which puckered the lung in areas. There were many minute air leaks, the source of the pneumothorax. The chyle appeared to rise from many areas over the mediastinal parietal surfaces and there was no intercommunication between the pleural spaces or localized disruption of the thoracic duct. Frozen section diagnosis of a malignant mesothelial cell growth was made. Although decortication was not successful, it was hoped that ligation of the thoracic duct would decrease the effusion of chyle. The thoracic duct was ligated just above the diaphragm and dissected proximally for 4 inches and excised. Three large thoracostomy tubes were left in the pleural space and attached to water-seal drainage bottles with suction. Complete re-expansion of the involved right lung could not be expected.

The pathologist reported, on permanent sections, malignant mesothelioma of the pleura, composed of uniform cells growing in sheets and strands, separated by a dense corrective tissue stroma. The cells showed vesicular nuclei, prominent nucleoli, and pale cytoplasm. In areas the cell boundaries were distinct. All tissue submitted showed the same type of infiltration.

The postoperative course was characterized by continued drainage of chylous fluid, and gradual deterioration in spite of a high caloric intake. Persistent air leaks, bilaterally, made it necessary to leave the thoracostomy tubes in both pleural spaces. Fractionated doses of nitrogen mustard in the pleural space did not decrease the amount of fluid draining daily. It continued to vary between 2,000 and 3,000 c.c. per day. Twenty days after thoracostomy, the patient quietly died.

Autopsy showed complete involvement of visceral and parietal pleura bilaterally with growth over the pericardium. The tumor extended into and below the diaphragm to involve the liver, the right adrenal, and the kidney. The mediastinum was extensively and deeply replaced by the growth.

DISCUSSION

The two uncommon features of the above case are: (1) the occurrence of spontaneous bilateral chylopneumothorax and, (2) the fact that it was due to a diffuse pleural mesothelioma.

Pleural mesothelioma will not be seen frequently in thoracic surgery and yet it is seen often enough to be kept in mind in the differential diagnosis of tumors of unknown types.[2] In a series of 60,042 necropsies, collected from various sources, pleural mesotheliomas were found in 43 cases or an autopsy percentage of 0.07 per cent. Stout[3] reported an accumulation of 72 cases in the laboratory of surgical pathology of Columbia University. Thirty-four were pleural and thirty-eight were peritoneal.

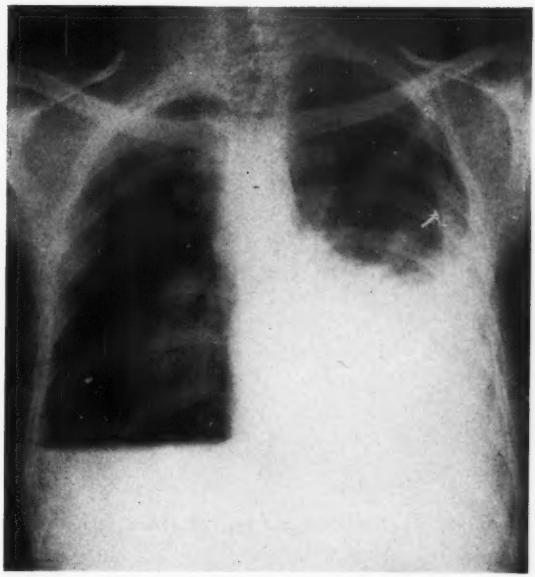

Fig. 1.—Bilateral chylopneumothorax

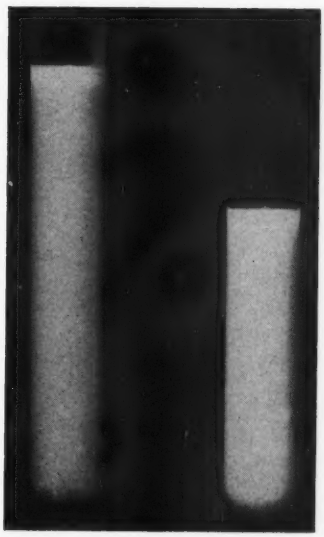

Fig. 2.—Fluid aspirated from the right chest.

Fig. 3.—Tumor of visceral and parietal pleura at operation.

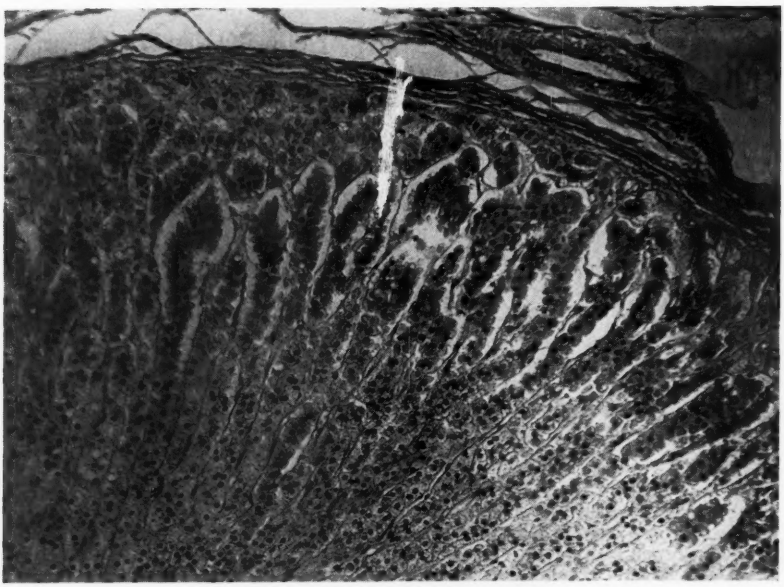

Fig. 4.—Microscopic picture showing strands of tumor cells having the "mosaic" pattern due to very distinct cell boundaries.

In 1950, Stout[4] reviewed the pathology of mesotheliomas and classified them in two main groups:

1. The diffuse variety which is highly malignant and microscopically shows epithelial elements forming tubular structures. These cells show the cultural characteristics of mesothelial cells and the ability to secrete hyaluronic acid.

2. Benign and malignant solitary fibrous mesotheliomas. These occur both in the pleura and in the peritoneum. They lack epithelial elements and consist of a bizarre stroma with spindle cells predominating. The benign type arises from a localized area of parietal or visceral pleural and projects freely out into the pleural space. The malignant type invades adjacent lung parenchyma and does not project freely into the pleural space. This variety has frequent mitosis, and variation in size and staining properties of the cells. The stroma is sparse in reticulum and collagen with areas of hemorrhagic necrosis.

Pleural mesotheliomas usually appear in the age group from 40 to 60 years. The ratio of males to females is approximately 2 to one. The symptoms and signs are variable and minimal, initially. This is an important feature of the clinical picture of both localized and diffuse mesotheliomas. Chest pain of a mild and intermittent nature is the most common complaint and may be elicited only on questioning. The first signs usually are those of a pleural effusion which rapidly re-accumulates following thoracentesis. It is usually bloody. Many times it is only after thoracentesis that a thickened pleura or a nodular density may be seen on x-ray. Any area of thickening along the mediastinal or lower pleural surfaces is suggestive. Pleural fluid cytology may reveal an increased number of mesothelial cells and many red blood cells.

The diagnosis is usually not made until exploratory thoracotomy is done. The histologic picture of mesothelioma is sufficiently characteristic to allow a certain diagnosis and this is of a practical importance in thoracic surgery, particularly concerning the localized type. This type when excised has a better prognosis than bronchogenic carcinoma. Therefore, it is important not to mistake it for an inoperable pleural metastasis from a bronchogenic carcinoma. There is no satisfactory treatment of the diffuse type. As far as can be determined every case reported and followed has been fatal.

In the case just reported it must be postulated that the cause of the nontraumatic chylothorax was compression, invasion, or occlusion of the thoracic duct by the tumor growth at multiple sites. This was not affected in the least by proximal ligation and excision of the thoracic duct. It was evident that the source of the air leak into the pleural space was from many holes in the visceral pleura and these were seen to be concentrated around the areas of puckering of the lung parenchyma by the tumor involving the visceral pleura. The commonest causes of nontraumatic chylothorax are (1) metastatic carcinoma involving the thoracic duct, (2) tuberculosis, (3) unexplained or miscellaneous causes, such as erosion of the duct by aortic aneurysm, occlusion of the duct by blood clot, lymphangiectasis of the duct, and thrombosis of the great vein of the neck.

SUMMARY

1. A case of spontaneous bilateral chylopneumothorax is reported. This was due to a diffuse pleural mesothelioma.

2. A review of the literature revealed no other similar case of spontaneous bilateral chylopneumothorax.

3. The literature revealed no other case of chylous fluid associated with a pleural mesothelioma.

4. This patient did not benefit from attempted decortication of the right lung and ligation of the thoracic duct.

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TRACHEOBRONCHIAL RESTS IN THE ESOPHAGUS

Their Relation to Some Benign Strictures and Certain Types of Cancer of the Esophagus

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THE occurrence of structures lined by tracheobronchial epithelium characterizes a group of congenital anomalies of the esophagus and sets them apart from other known anomalies of this organ. One may list the following as belonging to anomalies with tracheobronchial remnants[1, 2]:

1. Tracheoesophageal fistula. This is the commonest anomaly in this group, and the best known.
2. Cysts lined by bronchial epithelium. These may be either mediastinal,[3] or located in the wall of the esophagus.[4, 5]
3. Diverticula of the esophagus consisting of tracheobronchial tissue. When such a diverticulum projects beyond the esophageal wall it has been called a lung bud of esophageal origin.[6] In one reported case, the diverticulum was intramural and was termed an abortive tracheoesophageal fistula.[7]

It is apparent that all these anomalies can be regarded as the result of failure of the normal embryonal process of separation of the respiratory tract from the primitive esophagus. In the case of cysts, one might postulate that a tracheobronchial remnant, unconnected to either respiratory or intestinal tract, becomes distended by secretions. The diverticula represent remnants of tracheobronchial tissue with luminal continuity with the esophagus. Despite the simplicity and apparent adequacy of this explanation, other theories of formation of esophageal cysts and diverticula with tracheobronchial epithelium have been advanced and cannot be refuted.[8] These other explanations take two main forms. One is based on the fact that diverticula are normal in certain stages of embryonal development of the esophagus, and that such diverticula might become either pinched off or persist. Furthermore, the embryonic esophagus is lined by cylindrical epithelium, which might persist as such in the anomaly. The other explanation is that the esophageal epithelium is an irregularly vacuolated mass in the 8-week embryo and that anomalies may occur due to the failure of some of these vacuoles to coalesce with the normal esophageal lumen. Here, too, it would be postulated that these abnormal structures retain the embryonal type of epithelium. While at present none of these three theories can be either proved or disproved, it seems to us that the theory of failure of normal separation of the respiratory from the intestinal tract best accounts for the occurrence of these anomalies, and

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Received for publication Feb. 20, 1957.

especially for the occurrence of diverticula, such as the so-called lung buds and abortive tracheoesophageal fistulas, which consist not only of respiratory epithelium but also of cartilage, smooth muscle, and lymphoid tissue.

While the important role of congenital anomalies in the causation of clinical esophageal disease in infancy is universally recognized, the relationship of embryonal tracheobronchial rests to diseases of the adult esophagus is less well known. In the case of intramural diverticulum referred to above,[7] the congenital rest has been held directly responsible for the occurrence of a localized esophageal stricture. We would like to cite a second such case which was treated by local excision and end-to-end esophageal anastomosis.

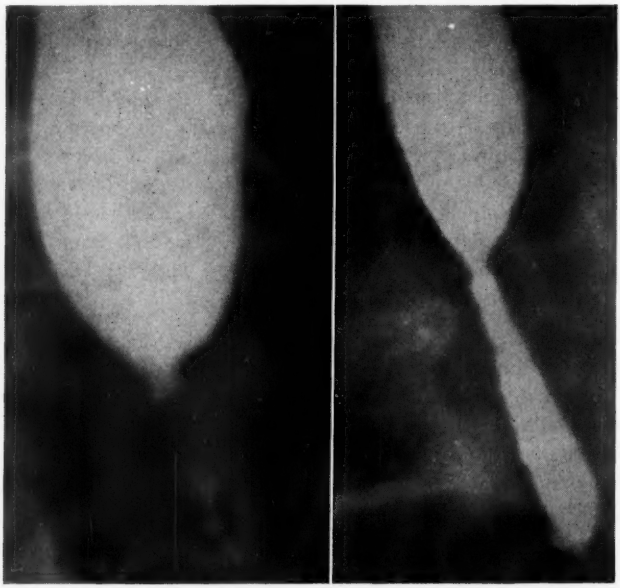

Fig. 1. Fig. 2.

Fig. 1.—Case 1. Esophagogram showing negative filling defect due to bolus of food impacted against esophageal stricture.

Fig. 2.—Case 1. Esophagogram showing stricture after disappearance of impacted bolus of food.

CASE REPORTS

CASE 1.—Hosp. No. 232104. S. F., a 57-year-old woman, was first admitted to the Jewish Hospital of St. Louis on April 15, 1953, with the chief complaint of dysphagia since childhood. She stated that in childhood she had had several episodes of complete inability to swallow foods and had had repeated endoscopic manipulations, with successful relief of dysphagia. Some dysphagia had persisted until ten years prior to admission, but during the decade before admission she had been quite asymptomatic. One week before admission, following a breakfast of bacon, eggs and toast, she developed complete dysphagia, with the sensation of food sticking at the level of the lower end of the sternum. The past history was entirely noncontributory.

Physical examination showed a well-nourished woman, who was in no distress. Blood pressure 140/90 mm. Hg, pulse 76, respirations 20, temperature 37° C. The only abnormal

findings were the presence of a moderately firm lymph node, measuring about 1.5 cm. in diameter, in the right jugular chain. This was said to have been present unchanged for many years. The liver edge was palpable 2 cm. below the right costal margin and was smooth.

Roentgenograms of the barium-filled esophagus showed a tapering, smooth stricture of the lower third of the esophagus and a negative filling defect thought to represent a bolus of food (Fig. 1). At esophagoscopy a smooth, tapering stricture was encountered at

Fig. 3.

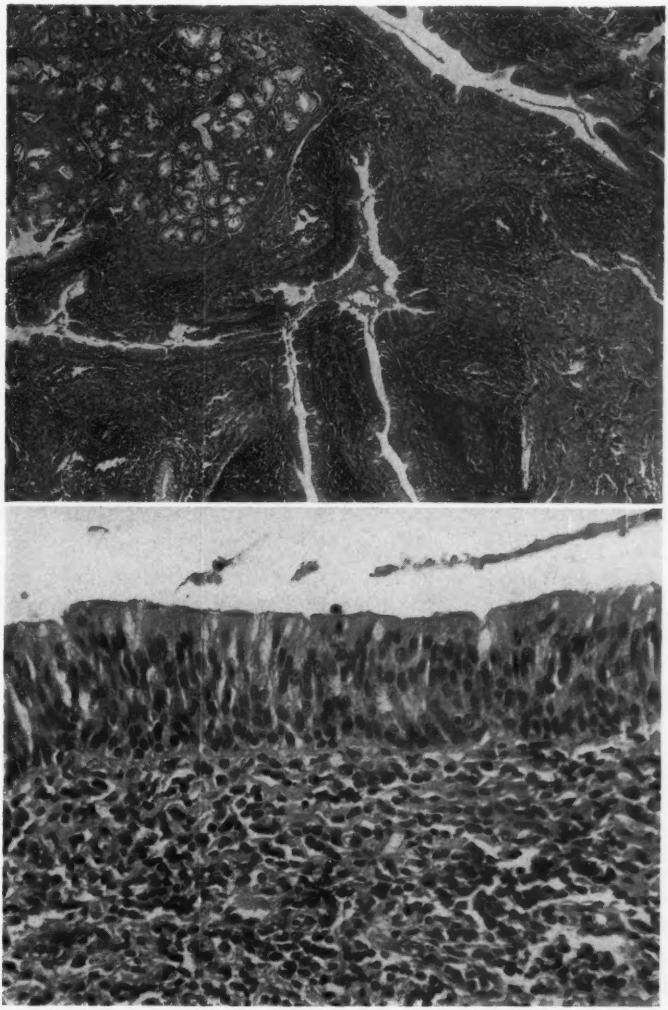

Fig. 4.

Fig. 3.—Case 1. Photomicrograph showing glandlike congenital rests deep in esophageal wall. Note associated lymphoid tissue and mucous glands (hematoxylin and eosin stain, ×35).

Fig. 4.—Case 1. Photomicrograph demonstrating cylindrical epithelium lining the congenital remnant (hematoxylin and eosin stain, ×250).

a distance of 42 cm. from the upper incisors. It did not permit passage of a No. 18 Fr. bougie. A second esophagogram on April 24, 1953, demonstrated that the bolus of food had disappeared and the stricture was now seen to be only about 1 to 1.5 cm. in length (Fig. 2). The patient was discharged on April 25, 1953, to return to the hospital at a later date for definitive therapy.

The patient was re-admitted to the hospital on May 19, 1953. Since discharge she had remained unable to take any but liquid foods. Physical examination was unchanged from the first admission and urinalysis, blood counts, and serologic tests were again within normal limits. On May 21, 1953, a left thoracotomy was done and the short esophageal stricture was found about 10 cm. above the diaphragmatic hiatus. A 3 cm. segment of the esophagus bearing the stricture was excised and esophageal continuity restored by end-to-end anastomosis. The patient's postoperative course was uneventful except for the development of some auricular premature contractions on the ninth postoperative day, which subsided after mild sedation. Esophagogram on the twelfth postoperative day showed a satisfactorily patent anastomosis and, at the time of discharge, on the fourteenth postoperative day, the patient was eating a soft diet.

Gross Description of Specimen.—Pathology No. 54126.* The specimen consists of a previously opened segment of esophagus measuring 2.5 cm. in length. The esophageal wall is firm and measures about 1.0 cm. in thickness. The esophageal mucosa is smooth except in the center of the specimen, where a distinct scar can be seen.

Microscopic Description.—The squamous epithelium is generally intact and in some areas appears thickened. The muscular layer of the esophagus is thickened due to extensive fibrosis. In one section there are two branching structures which have the appearance of very large glands or ducts (Fig. 3). However, they are located deep in the muscularis of the esophagus and they are lined by typical respiratory epithelium, such as is usually encountered in the trachea or major bronchi (Fig. 4). The lumen of these structures is not distended, but contains a small amount of mucus and debris. This would indicate a communication with the esophageal lumen, although this could not actually be demonstrated. The epithelium-lined structures were surrounded by lymphoid follicles and a marked chronic inflammatory reaction. Numerous mixed mucous and serous glands were also associated with these structures and emptied into them.

Comment.—While there is thus no question that benign obstructive lesions of the esophagus can be caused by changes initiated by the persistence of tracheobronchial rests in the esophageal wall, the concept that such rests might give rise to tumors of the esophagus has been advanced in the literature only once.[9] We believe the following case to be an example of a tumor originating in a tracheobronchial rest in the esophageal wall.

CASE 2.—Hosp. No. 560817. H. S., a 65-year-old man, was admitted to the Jewish Hospital of St. Louis on Feb. 1, 1956, because of dysphasia. He stated that he had had indigestion most of his adult life. This had consisted mostly of eructation, gas, and epigastric discomfort. However, previous office records of his referring physician indicated that he had also complained of some dysphagia for many years. He had had repeated roentgenographic studies of the upper intestinal tract, without abnormal findings. About two months prior to admission, he developed marked and definite dysphagia for solid foods, particularly meats, and had to begin washing foods down with water or other liquids. He localized the site of obstruction to the lower part of the sternum, where he also noted some discomfort with radiation to the left shoulder and scapula. The patient denied any weight loss. The past history was entirely noncontributory.

Physical examination at the time of admission showed the patient to be well nourished and well preserved for his age. The blood pressure was 170/80 mm. Hg. Temperature, pulse, and respirations were normal. Abnormal physical findings were limited

*We wish to thank Dr. Herman T. Blumenthal for his help with the pathological examination of this and the following case.

to the following: the heart extended to the midclavicular line in the fifth intercostal space. There was a soft apical systolic murmur and the second aortic sound was accentuated. Rhythm and rate were normal. The liver edge was palpable 3 cm. below the right costal margin on deep inspiration. It was smooth and nontender. There was a left indirect inguinal hernia.

Laboratory findings, including urinalysis, blood counts, fasting blood sugar, and blood urea nitrogen determinations, were all within normal limits. Roentgenography of the barium-filled esophagus showed an obstructing lesion of the esophagus at the junction of the middle and lower thirds. The lesion had the usual characteristics of a carcinoma (Fig. 5). Esophagoscopy was carried out on Feb. 3, 1956, and a fungating lesion, which virtually completely encircled the esophagus, was encountered at a distance of 30 cm. from the maxilla. A specimen taken for biopsy examination was thought to show epidermoid carcinoma.

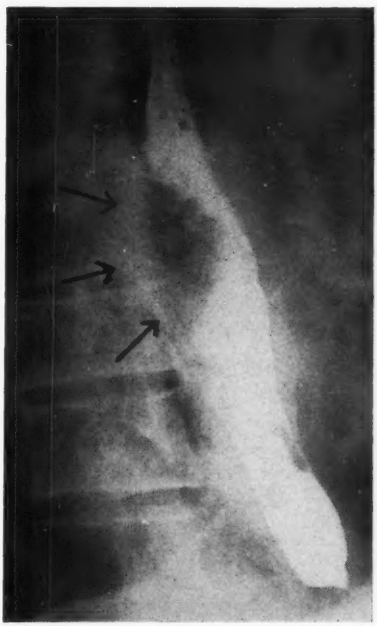

Fig. 5.—Case 2. Esophagogram. Arrows outline barely visible soft-tissue shadow caused by the tumor mass.

On Feb. 7, 1956, subtotal esophagectomy, using separate left upper abdominal and right posterolateral thoracotomy incisions, was carried out. The tumor occupied the middle third of the esophagus and esophageal continuity was restored by supra-aortic esophagogastrostomy. The patient's postoperative course was complicated by the development of auricular fibrillation on the day following operation. This responded promptly to digitalization and administration of quinidine. On the sixth postoperative day, the patient was found to be jaundiced. The icteric index was 25 units and bromsulfalein retention was 33 per cent. The jaundice gradually subsided and at the time of discharge on Feb. 23, 1956, the sixteenth postoperative day, the jaundice had cleared and the patient was eating soft foods without difficulty.

Gross Description of Specimen.—Pathology No. 66617. The specimen consists of a previously opened segment of esophagus measuring 13 cm. in length after fixation. About

2 cm. from the superior edge of the specimen there is a tumor with firm raised edges and deep central ulceration. It extends for a distance of 6 cm. in length, leaving about 5 cm. of uninvolved esophagus below the lesion. It virtually completely encircles the esophagus. Eleven lymph nodes were found in the mediastinal tissue attached to the esophagus.

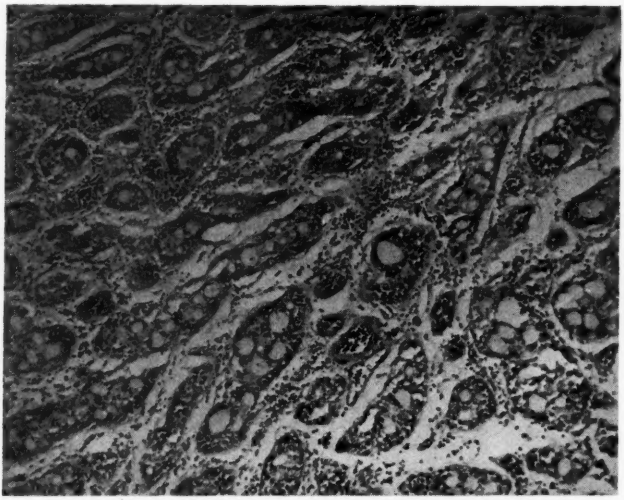

Fig. 6.—Case 2. Photomicrograph of main tumor mass showing characteristic cylindromatous appearance (hematoxylin and eosin stain, ×70).

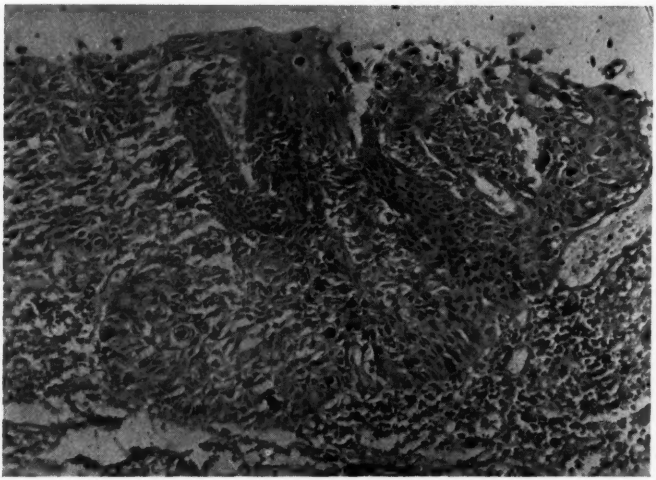

Fig. 7.—Case 2. Photomicrogram of in situ malignant change occurring in squamous epithelium overlying the tumor (hematoxylin and eosin stain, ×175; reduced ⅖).

Microscopic Description.—The tumor is made up of atypical epithelial cells of varying size. The cells have dense hyperchromatic nuclei with prominent nuclear membranes. These cells are arranged in small masses and cords, which often show distinct palisading of cells about a small central mass of basophilic material. The cell masses are separated

by a rather loose mature fibrous tissue stroma which has undergone marked basophilic change (Fig. 6). In some sections the squamous epithelium over the tumor remains intact and shows in situ malignant changes, consisting of loss of polarity, pleomorphism, and nuclear variation in size and staining quality (Fig. 7). However, there are no demonstrable areas of invasive squamous carcinoma, adenocarcinoma, or elements of stromal metaplasia as might be seen in a "mixed tumor." Periodic-acid Schiff stains failed to demonstrate any pink-staining material. Of the eleven lymph nodes, four contained metastatic tumor.

The in situ changes of the overlying epithelium were thought to be responsible for the erroneous diagnosis of squamous carcinoma made on the esophagoscopic biopsy specimen.

Comment.—The main point of interest in this case was the unusual microscopic structure of the tumor. It resembled closely the malignant cylindromatous tumors of the tracheobronchial tree.[10] It shared with them the finding of areas of more or less intact overlying epithelium, the lack of cornification of tumor cells, the finding of irregular cords and masses of tumor cells in a characteristic stroma, and the distinctive microscopic appearance of the tumor cells. A careful search of the literature has failed to turn up any previously published accounts of similar tumors originating in the esophagus. The lesion reported as an adeno-acanthoma, by McPeak and Arens,[11] deserves special consideration, because in the discussion of this case the authors relate this lesion to salivary gland tumors. The apparent similarity to salivary gland tumors rests primarily on the loose stroma which separates noncornifying tumor masses in their specimen. However, the authors' descriptions and illustrations demonstrate that the tumor is partly squamous carcinoma and partly glandlike in structure and thus qualifies for the term "adeno-acanthoma." In this regard it differs significantly from the tumor reported herein. The lack of reported cases indicates the rarity of malignant cylindromas of the esophagus such as our Case 2.

DISCUSSION

Speculation as to the origin of this tumor leads us to two hypotheses. Since the cylindromatous tumors of the tracheobronchial tree are believed to take their origin in the mucous glands of the tracheobronchial epithelium, one might assume that the tumor described by us arose in the mucous glands of the esophagus. In that case one would, however, expect to encounter such tumors with some degree of frequency as is true for the tracheobronchial cylindromatous lesions. Therefore we propose instead, an origin in a tracheobronchial remnant in the esophageal wall, such as was found in Case 1. The histogenesis of the tumor in the esophagus would then be identical to its more usual site in the tracheobronchial tree. This hypothesis would account for the extreme rarity of the lesion. It might also explain the long history of dysphagia with which our second patient presented himself. The prolonged dysphagia may have been caused by inflammation around a tracheobronchial remnant in the esophageal wall preceding the development of a carcinoma.

If our hypothesis that this carcinoma arose in a tracheobronchial rest in the wall of the esophagus is correct, we might expect to find other instances

of tumors characteristic of the tracheobronchial tree occurring in the esophagus. Interestingly enough, 2 cases reported by McKeown[9] fit in exactly with this concept. This author reported 2 cases of typical oat-cell carcinoma, indistinguishable from oat-cell cancer of the lung, occurring in the esophagus. She, too, felt that respiratory tract remnants were the likeliest sources of origin for these tumors. Her cases are also unique in the literature and they strengthen our belief that tracheobronchial remnants in the esophageal wall can cause not only benign strictures of the esophagus, but may also serve as starting points for tumors of the esophagus with the characteristics of primary lung tumors.

SUMMARY AND CONCLUSIONS

There is a group of congenital anomalies of the esophagus characterized by the presence of tracheobronchial rests. These remnants are thought to result from an abnormal separation of the primitive respiratory tract from the esophagus.

A second case of a benign stricture of the esophagus in an adult, due to inflammation around an intramural diverticulum of the esophagus lined by tracheobronchial epithelium, is presented. Treatment was by excision and end-to-end anastomosis.

A further case is reported in which a cylindromatous carcinoma with histologic features quite typical of such tumors in the tracheobronchial tree was found as a primary esophageal tumor. Such a lesion has not previously been reported. However, there have been 2 previous cases of oat-cell cancer identical to those commonly seen in the lung located in the esophagus. We believe that the occurrence of these tumors is best explained by assuming an origin in tracheobronchial rests in the esophagus.

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MORPHINE AND AMIPHENAZOLE FOR POSTOPERATIVE ANALGESIA FOLLOWING MAJOR THORACIC SURGERY

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SATISFACTORY analgesia for patients who have undergone major thoracic surgical procedures is not easy to achieve. The difficulty lies in the fact that morphine and its related compounds, though capable of providing satisfactory analgesia, have shown, owing to their depressant action on the respiratory and cough center, a tendency to produce atelectasis when used alone in sufficient dosage to produce really good analgesia.

The problem of postoperative atelectasis was discussed by Christie, Meakins, and Aronovitch,[1] who studied 23 patients treated with expectorants, postural drainage, bronchodilators, and antibiotics. They felt that pain in the chest on breathing, coughing, and moving was a major factor in the production of atelectasis following operations upon the chest. Bronchoscopic aspiration was necessary in 5 of the 23 patients.

Palmer and Sellick[2] studied a controlled series of 180 consecutive patients who had undergone abdominal surgery. They concluded that a deficient expulsive mechanism, a reduction in bronchial caliber, and qualitative or quantitative changes in the bronchial secretion were all important in the production of atelectasis. They obtained significant improvement in the incidence of postoperative atelectasis by using physiotherapy combined with bronchodilators.

The author of this paper felt that after major operations on the chest, the pain-factor assumed greater importance than in operations elsewhere, and was largely responsible for impaired pulmonary ventilation and ineffective cough. Thus, Björk and Hilty,[3] investigating the arterial oxygen tension of 17 patients after thoracoplasty or pulmonary resection, found an immediate postoperative decrease which they attributed to thoracic pain. This decrease was the greatest and lasted longest after thoracoplasty.

In this Thoracic Surgical Unit, it had been the custom to use morphine very sparingly after pulmonary resections, and not at all after thoracoplasties. In the latter case, pethidine was used in a dosage to be described later. Schaumann[4] described the analgesic effect of 100 mg. of pethidine, compared with 15 mg. of morphine as being about 0.15:1. Patients, particularly after thoracoplasty, often find it extremely difficult to cough because of the associated pain. For this reason it was thought that if morphine could be used in effective

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Received for publication Feb. 25, 1957.
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dosage, combined with some agent which would antagonize its respiratory depressant effect, the postoperative course of the patients would be more comfortable and the incidence of atelectasis might be minimized.

Shaw and Shulman,[5] in the treatment of a series of patients with inoperable malignant disease, showed that very large doses of morphine could be given in association with amiphenazole (2:4-diamino-5-phenylthiazole hydrochloride) without the occurrence of undue drowsiness or respiratory depression. They stated that amiphenazole, which was without toxicity, appeared to act as a nonspecific respiratory stimulant, especially when used parenterally. It was therefore decided to use morphine and amiphenazole for the postoperative analgesia of the patients in this investigation.

METHOD OF TRIAL

A consecutive series of 200 major thoracic operations was studied. The operations were carried out on 166 patients suffering from active pulmonary tuberculosis. The patients on whom the first 100 operations were carried out (hereafter known as the Control or Control Series) received the standard postoperative treatment, and those concerned in the remaining 100 operations (hereafter known as the Trial or Trial Series) received the treatment under investigation.

Treatment of Control Series.—After lung resections, the patients in this series received 100 mg. of pethidine, if necessary, on return to the recovery ward from the operating theater. On the night of operation, 1/6 or 1/4 grain of morphine was given, and thereafter 100 mg. of pethidine every 6 hours for three doses. On the night following operation, the morphine was repeated and, subsequently, three to four more doses of pethidine, 50 to 100 mg. were given if needed.

After thoracoplasty no morphine was given. Pethidine, 50 to 100 mg., was given every 4 hours to a total of 6 doses.

Treatment of Trial Series.—All patients received morphine as often as necessary to relieve pain. Usually this was not found to be necessary more frequently than every 4 hours. The dosage varied from 1/6 to 1/2 grain and was determined according to the response of the patient. Usually the women patients received 1/6 grain as an initial dose and the men 1/4 grain, but as the investigation progressed, the mental make-up of the patients was thought to be more important in determining dosage than considerations of sex or body weight.

The morphine was given intramuscularly and was mixed in the syringe on each occasion with amiphenazole solution. Initially 10 mg. of amiphenazole were used with 1/6 grain of morphine, and 15 mg. with 1/4 grain, but later 15 mg. of amiphenazole was a standard dose. Some patients required 30 mg.

A total of 974 doses of morphine with amiphenazole was given to the 80 patients who had 100 operations in this series.

Analysis of Material.—In the Control, the average age of all the patients was 27.7 years, that of the men 33.7 and of the women, 23. In the Trial, the

average age of all the patients was 34.6 years, that of the men 38.3 and of the women 29.9. There were 86 patients in the Control of whom 38 were men and 48 women, and 80 patients in the Trial of whom 45 were men and 35 women.

The average age of the 34 patients who underwent a total of 47 thoracoplasty operations in the Control was 34.5 years (range 18 to 52), and that of the 38 patients who underwent 57 such operations in the Trial 41.3 years (range 19 to 57).

In Table I, "one-stage thoracoplasty" refers to a planned one-stage procedure in which up to five ribs were removed. "First stage thoracoplasty" means an operation in which not more than 3½ ribs were dealt with at one time in a patient from whom it was planned to remove more ribs a fortnight later. Two-stage operations were, in general, carried out on patients in the older age groups and in those for whom an increased element of risk was thought to be present. The discrepancy between the numbers of first and second stage thoracoplasties in the same series is due to the fact that some patients undergoing two-stage thoracoplasty have had one operation excluded from the trial due to the consecutive nature of the series, and also because it was not thought advisable to proceed to a second stage in a few patients where this had been originally intended.

TABLE I

DESCRIPTION OF OPERATION	NUMBER OF OPERATIONS			
	CONTROL		TRIAL	
	NO.	TOTAL	NO.	TOTAL
One-stage thoracoplasty	15		8	
First stage thoracoplasty	16	47	24	57
Second stage thoracoplasty	14		20	
Resection and thoracoplasty	2		5	
Lobar resection	28		14	
Segmental and wedge resections	14	53	21	41
Pneumonectomy	11		6	
Others*	0		2	2

*The excision of a chronic tuberculous empyema with a chest-wall sinus in a man of 60, and a thoracotomy for closure of a postoperative bronchopleural fistula in a man of 25.

Palmer and Sellick[2] (1953), in the paper already referred to, felt that the quantity of sputum produced was relevant in their cases. In this Unit, routine measurement of sputum-volume is not made. There was a predominance of advanced cases in the Trial series which makes it unlikely that there was any significantly smaller volume of sputum produced by the patients in that series. In fact, the contrary might be reasonably expected.

It is necessary also to consider whether there were any relevant differences in anesthetic technique. All patients received a muscle relaxant, intratracheal intubation and controlled respiration in the usual way. Premedication was by either morphine and atropine or Omnopon and scopolamine. A few patients in the Trial series had premedication with promethazine, chlorpromazine and pethidine, and the author feels that these patients were more difficult to arouse postoperatively and required more amiphenazole than patients otherwise premedicated.

These facts indicate that the two series, though differing in the respects referred to, are not so constituted as to bias the results in favor of the treatment under investigation. Indeed the presence in the Trial Series of patients with more extensive disease, of an older age group and with a predominance of thoracoplasties, can, it is thought, be fairly regarded as a bias away from the Trial Series.

Method of Recording Results.—The following classification for the recording of degrees of atelectasis was used:

0—No atelectasis.
1—Slight or moderate atelectasis requiring no special treatment beyond the usual physiotherapy methods.
2—Atelectasis requiring special treatment other than bronchoscopy.
3—Atelectasis requiring bronchoscopy (these patients having had the treatment under 2 without effect).

Price Thomas[6] points out that the incidence of recognized postoperative atelectasis depends a great deal on the frequency with which clinical and radiologic observations are made. The patients in this investigation were x-rayed at least daily during the first two postoperative days, and about twice during the next three to four days. More frequent examinations were carried out as indicated, and the diagnosis of atelectasis was always made initially or confirmed by radiography.

In Grade 2 above, the special treatment referred to consisted originally of the intratracheal instillation of 50,000 units of crystalline penicillin in 2 ml. of sterile water. The injection was made via the cricothyroid membrane as for transcricoid bronchography through an intradermal bleb of local anesthetic. The method produced an explosive cough often successful in bringing about re-expansion of the lung. As this treatment was extremely unpleasant for the patient, it was decided, as the investigation progressed, to determine the effect of the intramuscular injection every 3 hours of 30 mg. of amiphenazole alone for five or six doses. This treatment came to supplant the use of intratracheal penicillin, and was used in the treatment of the last 5 cases in Grade 2 with complete success.

CASE REPORTS

The following two case histories are illustrative of the treatment and results obtained.

CASE 1.—A man of 33 years of age had persistent cavitation in the upper lobe of the right lung for which a right upper lobectomy was carried out on May 9, 1956. Postoperatively he had two doses of 1/6 grain of morphine and three of 1/4 grain of morphine, each combined with 15 mg. of amiphenazole, between 2:10 P.M. on May 9, 1956, and 9:00 A.M. on May 10, 1956. He was a heavily built man and was found to have a low threshold to pain. Because of this, the dose of morphine was increased to 1/3 grain, with 30 mg. of amiphenazole, at 1:00 P.M. on May 10, 1956. Analgesia was produced for about 3 hours only and, at 5:00 P.M. on May 10, 1956, the dose of morphine was increased to 1/2 grain with 30 mg. of amiphenazole. This produced satisfactory analgesia for 7 1/2 hours, and, owing to the freedom from pain the patient was able to cough well. Subsequent analgesia

was obtained by ⅓ grain of morphine and 15 mg. of amiphenazole which was repeated 5 times, and ¼ grain with 15 mg., repeated twice. The postoperative course was entirely uneventful and there was no evidence of atelectasis at any time.

CASE 2.—A man of 46 years of age came to operation on June 11, 1956, for a chronic left sided empyema of about eighteen months' duration which was a sequel to a left spontaneous pneumothorax complicating cavitated pulmonary tuberculosis. At operation the empyema was excised and a resection of the left upper lobe carried out. This was extremely difficult as mobilization of the lung was hindered by the extensive pleurisy. It was anticipated that difficulty might be encountered with this patient's postoperative course. He received 16 doses of ⅙ grain of morphine, 12 of which were given with 10 mg. of amiphenazole, and the remainder with 15 mg., from 2:45 P.M. on the day of operation to 11:30 P.M. on June 15, 1956. Breath sounds at the left base were poor, and x-ray, on June 15, 1956, showed an opaque left hemithorax due to atelectasis. He was given 30 mg. of amiphenazole alone every 3 hours for 5 doses and then every 4 hours for a further 5 doses, intramuscularly. During the next two days the breath sounds returned and serial x-rays demonstrated satisfactory expansion of the residual lower lobe. The subsequent course was uneventful.

RESULTS

The gross results of the investigation are presented in Tables II and III.

TABLE II

GRADE OF ATELECTASIS	NUMBER OF OPERATIONS	
	CONTROL	TRIAL
Grade 0	67	76
Grade 1	18	17
Grade 2	8	7
Grade 3	7	0
Total number of operations	100	100

TABLE III

OPERATION	GRADE OF ATELECTASIS									
	CONTROL					TRIAL				
	0	1	2	3	TOTAL	0	1	2	3	TOTAL
Thoracoplasty, including resection and thoracoplasty	27	11	3	6	47	40	12	5	0	57
Resection, including pneumonectomy	40	7	5	1	53	35	4	2	0	41

DISCUSSION

Table II shows that 7 bronchoscopies were found to be necessary in the Control, and none in the Trial, an incidence of 7 per cent and 0, respectively. The standard error of the two proportions is 2.598 per cent, and the observed incidence of 7 per cent is therefore 2.69 times this error.

In Table III, 6 bronchoscopies were found to be necessary among the 47 thoracoplasties in the Control, and none among the 57 in the Trial, an incidence of 12.7 per cent and 0, respectively. The standard error of the two proportions is 4.594 per cent and the observed incidence of 12.7 per cent is 2.78 times this error. In the other cells in Table III there is no significant difference between the Control and the Trial.

Clinically, the treatment made a good impression. The patients were undoubtedly given a much more comfortable postoperative course, particularly after thoracoplasty, and it is noteworthy that 6 of the 7 bronchoscopies in the Control followed thoracoplasty operations. This will be compatible with the experience of most thoracic surgeons. Vomiting, which could be unequivocally ascribed to the morphine, and which led to its discontinuance, was met with in only 2 patients.

SUMMARY

A consecutive series of 200 major thoracic surgical operations for pulmonary tuberculosis has been studied, and a controlled trial of the effectiveness of morphine and amiphenazole, administered simultaneously, in the production of effective postoperative analgesia without danger of atelectasis, has been made.

It is concluded that morphine-amiphenazole therapy is superior to the other routine treatments described, both in the production of satisfactory postoperative analgesia, and in the avoidance of atelectasis of a degree sufficient to require bronchoscopy. In this series the major incidence of postoperative bronchoscopy occurred after operations involving a thoracoplasty, and it is in this group that the treatment described has been found most effective.

It is suggested that morphine with amiphenazole may be of value after extrapulmonary thoracic operations and high abdominal operations, following which atelectasis is regarded as a likely complication.

The use of amiphenazole for the treatment of established atelectasis is described, and it is suggested that this treatment is always worthy of trial before resort is made to bronchoscopic aspiration.

The writer wishes to thank Dr. J. R. Edge for helpful encouragement, and Miss I. Dingwall Fordyce (by courtesy of Professor R. E. Lane) for statistical advice. The operations were carried out by Mr. J. S. Glennie, Mr. P. Jewsbury, Mr. H. A. Daniels, and Mr. N. C. Scott.

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PLEURECTOMY FOR RECURRENT SPONTANEOUS PNEUMOTHORAX

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OBLITERATION of the pleural space is generally recognized as an important feature of the definitive therapy for spontaneous pneumothorax. Obliteration both by closed and open surgical procedures has been incomplete and unsatisfactory in many instances.[1-8] Seeking a solution, we have adopted excision of the parietal pleura to promote fixation of remaining lung tissue to the chest wall. In August, 1951, we treated our first in a series of patients for recurrent spontaneous pneumothorax by thoracotomy, lung repair, and partial parietal pleurectomy. This report includes an additional 13 patients who have undergone fourteen such procedures since that time.

Surgical removal of normal parietal pleura has been proposed for several pathologic conditions. Barrett and Daley,[9] in 1949, suggested bilateral excision of the upper one half of the parietal pleura as an alternate procedure for the technically inoperable patient with tetralogy of Fallot. Blalock[10] agreed that the rationale of increasing the blood supply to the lung by pleurectomy in selected patients had merit. Crenshaw and Rowles[11] have employed parietal pleurectomy in order to revascularize the degenerated, atrophic lungs of patients with pulmonary emphysema. Ellis and Carr,[12] in 1954, suggested pleurectomy be tried on selected patients with spontaneous pneumothorax in whom surgery is indicated. Gaensler[13] recently reported the first series of patients with recurrent spontaneous pneumothorax in whom parietal pleurectomy was performed as an important feature of therapy. Good results were reported in 7 patients, with excellent fusion between the lung and chest wall. He recommended pleurectomy for two groups of patients—those found to have diffuse lung surface involvement, and those with little or no visible gross pathologic lesions at the time of thoracotomy.

In August, 1951, we performed a thoracotomy on a patient with recurrent spontaneous pneumothorax. Lung repair was carried out in the usual manner, by excision and suture of multiple blebs over the apex of the left upper lobe. At this time it occurred to one of us (P.W.G.) that excision of the apical parietal pleura in the region of the disease process would be a feasible method of obliterating the pleural space. This was effected with a good result. In a second patient with bilateral, giant, emphysematous bullae, surgery was indicated for decompression of contiguous good lung tissue. In January, 1952, right thoracotomy, apical and posterior segmental resection, and parietal pleurectomy were done. At a second stage, in July, 1952, left thoracotomy, left

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Received for publication Feb. 25, 1957.

upper lobectomy, and parietal pleurectomy were performed. Following the second procedure, the patient experienced considerable improvement in respiratory function; over the past four years the patient has continued to be asymptomatic and the lungs are shown to be clear by chest roentgenograms. Consequent to the experience with these 2 patients, excision of the parietal pleura has been instituted as a component part of our surgical treatment for recurrent spontaneous pneumothorax and for giant bullous emphysema. We feel that lung repair with pleurectomy, followed by continuous, active, and complete re-expansion of the lungs is the most reliable method for obliteration of the pleural space. The careful management of the pleural space in the early postoperative period, to ensure complete expansion and fusion of the lung to the chest wall, is as essential as the pleurectomy.

Since January, 1951, 108 patients admitted to this hospital have been treated for 122 episodes of spontaneous pneumothorax. Among these patients, 17 had three or more episodes of spontaneous pneumothorax treated here or elsewhere. Criteria for surgical intervention included recurrence of spontaneous pneumothorax (three or more episodes); demonstration of cystic areas within the lung fields by chest roentgenograms following spontaneous pneumothorax (one or more episodes); or persistent air leak with incomplete lung expansion (ten days or longer) during treatment of the acute episode. Twelve of our patients have been subjected to thoracotomy. Two additional patients operated upon elsewhere by one of us are added to make the total 14.

The indications for surgery are listed in Table I. Ten patients had recurrent spontaneous pneumothorax without roentgenographic evidence of lung field abnormality. In one patient (Case 10) surgery was advised following a single episode of spontaneous pneumothorax associated with cystic areas of the lung field demonstrable on the chest roentgenogram. Another patient (Case 7) was treated surgically after two episodes of spontaneous pneumothorax who also had roentgenographically demonstrable cystic areas of the lung field. Bilateral surgery (Case 11), done in two stages, was indicated for alternating spontaneous pneumothorax associated with bilateral cystic areas of the lung fields demonstrated by chest roentgenograms. In another instance (Case 13), surgery was accomplished during the first acute episode because of a persistent air leak with incomplete lung expansion during intercostal catheter suction therapy. Our total experience with parietal pleurectomy during this 5-year period further includes 6 other patients treated surgically for giant bullous emphysema. Two of these patients underwent bilateral surgical intervention. In all, 20 patients have had twenty-three operations in which parietal pleurectomy was considered an important component of the surgical procedure.

The surgical removal of normal parietal pleura is begun along the cut margins of the pleura, at the edges of the thoracotomy incision. The proper tissue plane is developed by blunt dissection and the pleura stripped in a single large sheet away from the chest wall. The superior dissection is carried over the apex of the chest and down along the mediastinal surface to the azygos vein on

TABLE I. CLINICAL DATA

CASE	AGE	SEX	SIDE OF PNEUMOTHORAX			GROSS SURGICAL PATHOLOGY	SURGICAL PROCEDURE	POSTOPERATIVE COMPLICATIONS	PRESENT STATUS
			R	L	TOTAL				
1.	26	M	0	3	3	Two blebs, apex LUL	Lung repair, apical pleurectomy	None	Unknown
2.	20	M	0	3	3	Multiple blebs LUL and basal segments LLL	Lung repair, pleurectomy	None	Unknown
3.	21	M	3	0	3	Multiple blebs, apex RUL	Lung repair, pleurectomy	Air leak (7 days)	Asymptomatic (24 mo.)
4.	22	M	0	3	3	Multiple blebs, apex LUL	Lung repair, pleurectomy	Rt. spontaneous pneumothorax (day of surgery)	Asymptomatic (22 mo.)
5.	40	F	0	3	3	Necrotic visceral pleura plaque, apex LUL	Lung repair, pleurectomy	None	Unknown
6.	41	F	4	0	4	Paper white, thin unelastic appearance RUL	Lysis of adhesions, pleurectomy	None	Asymptomatic (18 mo.)
7.	21	M	2	0	2	Multiple bullae and blebs RUL	Lung repair, pleurectomy	Apical pneumothorax obliterated 4 mo. after surgery	Left spontaneous pneumothorax 7 mo. after surgery, rt. asympt. (15 mo.)
8.	24	M	4	0	4	Single large bullous apex RUL	Lung repair, pleurectomy	Atelectasis; left spontaneous pneumothorax immed. postop.	Left spontaneous pneumothorax, 3x, rt. asymptomatic (14 mo.)
9.	19	M	3	0	3	Multiple pleural scars, apex RUL	Pleurectomy	None	Asymptomatic (13 mo.)
10.	34	M	1	0	1	Multiple bullae and blebs, apex RUL	Lung repair, pleurectomy	None	Asymptomatic (10 mo.)
11.	21	M	1	2	3	Multiple bullae and blebs, apex LUL Multiple blebs, apex RUL	Bilateral surgery, lung repair pleurectomy	Left—none Right—air leak (9 days)	Asymptomatic (6 mo.)
12.	37	M	1	2	3	Multiple blebs, apex LUL	Lung repair, pleurectomy	Small residual pneumothorax, apex	Rt. spontaneous pneumothorax (6 mo.)
13.	21	M	0	1	1	2 mm. peripheral air leak, apex LUL	Lung repair, pleurectomy	None	Asymptomatic (5 mo.)
14.	30	F	4	0	4	Multiple blebs, apex RUL	Lung repair, pleurectomy	None	Asymptomatic (5 mo.)

the right, or aortic arch on the left, where the pleura is excised. A similar dissection of the pleura is carried out inferiorly to the diaphragmatic reflection. No attempt is made to remove the parietal pleura of the diaphragm or pericardium. At the conclusion of the operation, and prior to closure of the incision, large thoracotomy drainage tubes are carefully placed for continuous postoperative suction.

The postoperative complications are shown in Table I. Because of the large raw surface produced by pleurectomy, careful hemostasis must be obtained and postoperative hemorrhage watched for. Blood should be available for administration during and following the operative procedure. Troublesome bleeding has not been a complicating feature in these patients; however, whole blood replacement of an estimated average operative loss of 1,500 to 2,000 c.c. is routine. The complications encountered are not directly attributable to pleurectomy. They have resulted more frequently because of inadequate control of air leaks. The postoperative course was entirely uneventful following nine operations. A somewhat prolonged air leak was present in two instances (Cases 3 and 11) but recovery was uneventful. One patient (Case 4) developed a minimal spontaneous pneumothorax of the unoperated side of the chest the day of surgery; treatment was not necessary and recovery not remarkable. In two instances (Cases 7 and 12), a small, residual postoperative apical pneumothorax was encountered. Of the 15 operations performed only one resulted in a seriously complicated course (Case 8). Immediately following surgery, this patient developed partial atelectasis of the operated lung. Obliteration of the pleural space was therefore incomplete. Bronchoscopy and thoracentesis were required as corrective measures. Further, this patient also had a spontaneous pneumothorax of the unoperated lung shortly after surgery which contributed to his morbidity. One additional, more serious complication worthy of mention was encountered in the group of patients treated for bullous emphysema. Of the 6 patients selected as candidates for surgery, eight operations were done (two bilateral thoracotomies) in which pleurectomy again was an important feature of the surgical procedure. In one of these patients a postoperative bronchopleural fistula developed. This was followed by a *Staphylococcus aureus* empyema. After prolonged hospitalization the patient responded to repeated thoracentesis and Terramycin therapy.

The present status of these patients is likewise listed in Table I. There have been no postoperative recurrences of spontaneous pneumothorax. Eight patients are asymptomatic. One of these (Case 11) required bilateral surgery. In three instances (Cases 7, 8, 12), the postoperative lung is asymptomatic and stable by chest roentgenogram; however, one or more episodes of spontaneous pneumothorax of the unoperated lung have occurred. These patients may require a second-stage surgical procedure on the remaining unstable lung at some future date for complete relief from this condition. Because of a shifting military population, 3 patients (Cases 1, 2, 5) have been lost to follow-up evaluation. However, they were asymptomatic during a 6- to 12-month period of observation.

DISCUSSION

There is a general agreement on the indications for surgery in patients with spontaneous pneumothorax. The most common is recurrent spontaneous pneumothorax, because of the morbiditiy and associated economic loss to the patient. We have limited our selection of patients primarily to those having had three or more episodes of proved spontaneous pneumothorax; 10 of the tabulated patients are in this category. MacQuigg[7] makes a strong plea for earlier surgery for these patients on the basis that gross pathology is usually present and the recurrence rate is high. The second most frequent indication for operative intervention in this experience was one or more episodes of spontaneous pneumothorax with associated cystic areas demonstrated by chest roentgenograms. Three of our patients are in this category. This was the indication for bilateral surgical intervention in one instance (Case 11). Finally, in one patient (Case 13), thoracotomy was necessary because of an uncontrolled air leak during treatment of the first episode of spontaneous pneumothorax. Hemopneumothorax of the spontaneous variety is also considered by some an indication for thoracotomy. The mortality from spontaneous pneumothorax complicated by hemothorax has been estimated to be about 20 per cent.[14] However, we have treated 3 patients with spontaneous hemopneumothorax of moderate degree (500 to 1,000 c.c. of blood removed by intercostal catheter) without resorting to thoracotomy. Possibly the hemothorax produced pleural symphysis; at least there have been no recurrences in these 3 patients. Although we have had no experience in chronic pneumothorax complicated by a fibroplastic membrane which encases the lung, we feel that this condition is also an indication for surgery. This latter condition should occur less and less frequently as more and more physicians become aware of the merits, for the acute episode, of active and immediate re-expansion of the lung by intercostal catheter, water-seal suction drainage of the pleural space.

The predominant gross surgical pathologic lesion found in our patients was the presence of multiple blebs and bullae localized to the apical lung surface. Such was the case at ten operations. In one instance (Case 2), a more generalized distribution of multiple blebs was found. The lung surface of one patient (Case 5) appeared grossly normal with the exception of a single, small area of necrotic visceral pleura at the apex. In another patient (Case 9), the lung surface again appeared grossly normal except for several, small, healed, pleural scars without associated surface blebs. In still another instance (Case 6), the entire upper lobe surface appeared paper-white, thin, and unelastic, witih numerous adhesions indicative of fibrotic change. Finally, in one patient (Case 13) there was a single, 2 mm. air leak in the apex with rolled in fibrotic edges. These characteristic findings of blebs, bullae, and parchment-like changes are similar to some of the gross changes of pulmonary emphysema. They are suggestive of peripheral pulmonary ischemia. Whether this represents a primary or a secondary vascular insufficiency remains to be determined.

One of the fundamental objectives of definitive surgical treatment for recurrent spontaneous pneumothorax is the elimination of the pleural space, which we feel is best accomplished by excision of the parietal pleura. The increased vascularity of the peripheral lung produced by this procedure may be of considerable future benefit to the patient. The incidence of spontaneous pneumothorax in young people appears to be on the increase. One of us (P.W.G.) feels that this probably represents a precursor to the pulmonary changes recognized later in life as emphysema. Spontaneous pneumothorax may be an early clinical manifestation of an atrophic, degenerative alteration of the lung parenchyma. In the beginning this may be localized to an area of involvement, but ultimately destined to progress to more extensive or diffuse pulmonary disease. If they are the result of diminished pulmonary vascularity, progression of these fundamental pathologic changes might be prevented by early parietal pleurectomy. This operative procedure seems to provide an increment of blood supply to the lungs. Therefore, parietal pleurectomy is indicated for the patient with localized pathology as well as the more obvious instance of the patient with diffuse lung surface involvement, or the more rare patient without gross disease demonstrable at surgery. This concept will probably have to remain a theoretical one for years until such time as long-term, follow-up evaluation of patients is possible.

SUMMARY

We have presented a series of 14 patients in whom partial, parietal pleurectomy has been an important feature in the definitive surgical treatment for recurrent spontaneous pneumothorax. The surgical removal of the parietal pleura over the chest wall and superior mediastinum, except for the diaphragmatic and pericardial reflections, as described, is usually not difficult. A pleurectomy limited to excision of the apical parietal pleura, as was carried out in the first patient, we now consider inadequate. The more complete operation was done fourteen times in 13 patients. Pleurectomy is designed to produce two significant changes for the patient: (1) to obliterate the pleural space and prevent recurrence, and (2) to provide revascularization of the peripheral portion of the lung where pathologic changes are usually most marked.

In the majority of patients subjected to thoracotomy for recurrent spontaneous pneumothorax, gross pulmonary pathogenicity, in the form of blebs or bullae, is demonstrated either in localized areas or generalized over the lung surface. If this is a precursor of later pulmonary emphysema, with primary or secondary vascular changes, pleurectomy should be considered more frequently than it has been in the past.

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THE SURGICAL MANAGEMENT OF CONGENITAL BIFID STERNUM WITH PARTIAL ECTOPIA CORDIS

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CONGENITAL bifid sternum associated with partial ectopia cordis is an infrequently encountered anomaly. However, when present this malformation may present a difficult problem in reconstruction. Recently, attention has been directed toward the surgical management of this condition and several procedures have been advocated. In 1947, Burton[1] reported his experience with 2 cases in which grafts of costal cartilage were used to close the defect. Later Maier and Bortone[2] recommended direct surgical closure of the munication is to report an operative technique employed to repair a large defect in infancy and reported a successful case. Two other instances of operation have been reported subsequently.[3, 4] The purpose of the present comsternal defect associated with partial ectopia cordis in a child, 2 years of age.

CASE REPORT

T. V., a 28-month-old boy, was admitted to the Johns Hopkins Hospital on Sept..25, 1956, with the history of a bulge in the sternal area since birth. The family history was negative for congenital defects and the child was the result of a normal, full-term pregnancy. The early development was unremarkable except for moderate difficulty in swallowing during the first month of life. The child had been examined first at the age of nine months. At that time he appeared well developed and was in no distress. A triangular defect was noted in the sternal area and the two sides of the chest were connected by a small bridge, one centimeter in length at the xiphoid process (Fig. 1, *A*). The distance between the clavicles was 6 cm. The pulsations of the aorta were visible beneath the skin overlying the defect and, with crying, a marked herniation of mediastinal contents occurred. The remainder of the physical examination was within normal limits. The laboratory findings were not remarkable. At that time it was elected to defer operation until a later date.

On the second hospital admission the history revealed that the child had been asymptomatic and had developed normally during the intervening period. The previously described sternal defect was slightly larger than at the first examination. The child's family was increasingly concerned about the defect and were alarmed at the marked herniation which occurred through it with crying or coughing.

On Oct. 2, 1956, operation was performed. The child was anesthetized with cyclopropane and a midline vertical incision was made from the lower neck to the xiphoid process. Skin flaps were dissected from the underlying pericardium and reflected beyond the sternal bands (Fig. 2). The sternal bands were separated from their posterior attachments to the pleura, and an encircling suture of braided silk was placed temporarily around the sternal bands and approximated. This reduced the size of the defect to approximately

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Received for publication Feb. 28, 1957.
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Fig. 1—*A*, Preoperative photograph demonstrating the sternal defect with partial ectopia cordis. With crying, a marked herniation of mediastinal contents is apparent. The two sides of the chest are connected anteriorly by a small bridge at the xiphoid process.

B, Lateral view showing sternal defect during inspiration. The point of union of the two sternal bands is seen at the xiphoid process.

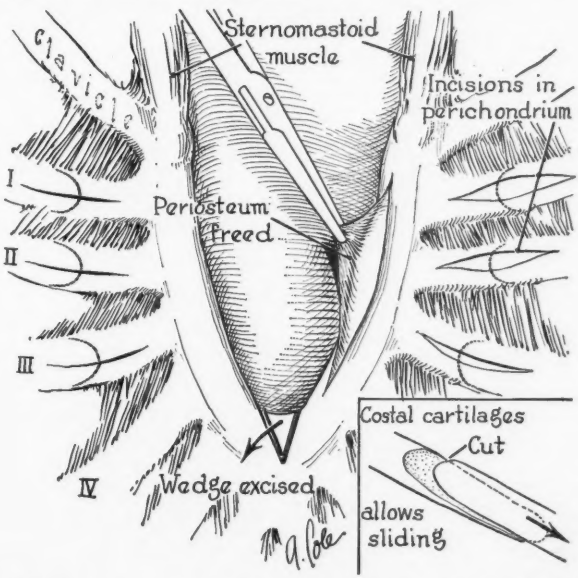

Fig. 2.—Illustration of the defect after dissection of the skin flaps. The sternal bands are shown and the periosteum is being freed from the anterior surface. Incisions have been made into the perichondrium of the first, second, third, and fourth costal cartilages. The inset illustrates the incisions in the costal cartilages.

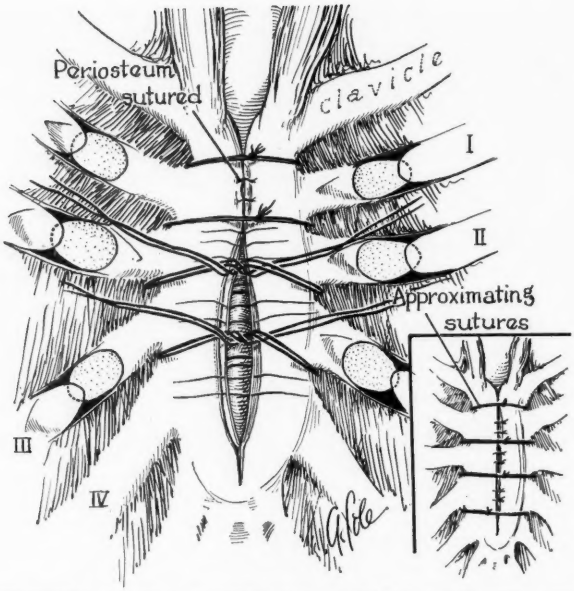

Fig. 3.—Illustration showing approximation of sternal bands with encircling sutures following the oblique incisions in the costal cartilages. Multiple fine silk sutures are shown in the periosteal flaps. The inset shows the final appearance.

Fig. 4.—*A*, Postoperative photograph. *B*, Postoperative photograph (oblique).

one half of the original dimensions and brought into clear view the attached costal cartilages on either side. The costal cartilages of the first, second, and third ribs were exposed and the perichondrium was reflected (Fig. 2). An oblique incision was made in the costal cartilages of ribs one, two, and three which allowed sliding, as illustrated in the inset of Fig. 2. The periosteum was incised along the medial border of each of the sternal bands and was reflected anteriorly. Three sutures of chromic catgut were then placed about both sternal bands and were tied. This completely approximated the bands and obliterated the defect. The periosteal flaps of the sternal bands were then carefully sutured with fine silk uniting the sternum in the midline (Fig. 3). At this point the suprasternal notch was approximately in its normal position. After closure of the defect the anesthetist was unable to detect any change in the pulse, blood pressure, or color of the face or upper extremities. In addition there was no increased resistance to inflation of the lungs through the endotracheal tube. The child made an uneventful postoperative recovery and has since remained well. The chest now appears essentially normal and the chest wall is solid (Fig. 4, *A* and *B*).

DISCUSSION

Congenital failure of sternal fusion is best explained by a consideration of the embryology of the normal sternum. The work of Hanson in tracing the origin and development of the sternum in man and a number of animals has added much to our understanding of congenital bifid sternum. He showed that the sternum originates independently of the ribs as two longitudinal bands in the anterior thorax. These bands are present in the 6-week embryo. At this time there is a space between the two bands, and by the ninth week the two bands have fused in the midline with obliteration of the defect. The bands also fuse laterally with the costal cartilages. It becomes apparent that in the rare instances of congenital bifid sternum there is a failure of the midline union of these two sternal bands. A defect results and through it the pericardium may herniate, being covered only by subcutaneous tissue and skin. This condition has been termed either congenital bifid sternum with partial ectopia cordis or failure of sternal fusion with pericardial herniation.

In 1947, Burton reported his experience in the surgical treatment of 2 cases of this type. His first patient was a child of 7 weeks who had recurrent attacks of cyanosis and dyspnea. A block of cartilage was removed from the costal margin and placed over the defect and sutured in place. The second defect was in a 12-year-old girl with a similar condition. The same operative procedure was performed as in the first case and in both the cosmetic result was only moderately good.[1] In 1949, Maier recorded the case of a 6-week-old infant in which he performed a primary surgical closure.[2] In this instance, the two sternal bands were approximated by direct suture and a good result was obtained. During the discussion of Maier's paper, Klassen reported a similar case which he had attempted unsuccessfully to repair.[3] The most recent report of a surgical repair is that of Longino and Jewett.[4] They discuss an infant upon whom they operated at the age of 6 days. The defect was closed by primary approximation of the two sternal bands with encircling sutures with a successful result.

It appears from the recent literature that the recommended management of congenital bifid sternum with partial ectopia cordis is early operation and

primary union of the sternal bands. If operation is performed during infancy, there is no necessity for the use of tissue grafts or plastic materials in the repair of the malformation. In the child reported in this communication the defect was not attacked surgically until the age of two. Nevertheless there were no difficulties encountered at the time of operation and the defect was managed by primary closure. The ease with which this was accomplished is thought to be due to the operative technique employed. A vertical incision was made over the center of the defect and skin flaps were reflected to each side. The sternal bands were freed of their posterior pleural attachments and the costal cartilages which attach to the bands were exposed (see Fig. 2). These costal cartilages were then divided in an oblique manner as illustrated in the inset in Fig. 2. This allowed them to slide and reduce the tension without interfering with the stability of the anterior chest wall. This sliding-type of repair permitted easy approximation of the two sternal bands in the midline by the use of encircling sutures. To strengthen further the midline union, periosteal flaps were reflected anteriorly from each of the sternal bands and were approximated with interrupted sutures. An instance of separation of the sternal bands in the late postoperative period has previously been reported[3] and it is probable that this complication can be prevented by the separate closure of the periosteum.

Despite previous discussions in the literature regarding the difficulty, if not impossibility, in obtaining primary closure of these sternal defects after infancy, the experience reported in the present communication suggests that operation can be simple and effective when it is necessary to perform it at a later date. By use of the sliding costal cartilage technique no difficulty was encountered in the correction of the deformity. In the event a child does not present for surgery until several years of age or in instances in which operation is deferred for other reasons, the operative technique discussed here may be employed with satisfactory primary closure without resort to the use of plastic prostheses or tissue grafts.

SUMMARY

The surgical management of congenital bifid sternum with partial ectopia cordis has been discussed. It is noted that there is general agreement that, when possible, the preferred management is direct operative closure in early infancy. An operative technique is described which permits successful direct closure of the malformation at a later age without the use of grafts or prosthetic materials.

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COARCTATION OF THE AORTA PROXIMAL TO BOTH SUBCLAVIAN ARTERIES

Case Report of a Six-Year-Old Girl

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AS FAR AS we are aware, coarctation of the aorta proximal to both subclavian arteries has not been described previously. One of our patients presented this anomaly, combined with a right aortic arch and a left-sided descending aorta. Preoperatively, no pulse was palpable in either the arms or the legs, whereas the carotids were hyperactive. This case seems to be of interest from the point of view of the surgical technique as well, since it was possible to correct the retroesophageal course of the aorta, so that, postoperatively, it ran in front of the trachea.

Coarctation of the aorta may be present from the middle of the arch to the bifurcation, but in 98 per cent of the cases it is located at the first segment of the descending aorta, immediately distal to the arch.[14] According to Goodson[11] and Mustard and co-workers,[18] coarctation of the aorta is found in one of 1,500 necropsies. A conception of the incidence of this malformation in infants and young children with congenital heart disease is obtained from a survey made by Boeson and Vendel.[10] In 1,137 autopsies on children under 4 years old with congenital heart disease, they found 221 cases of coarctation of the aorta, that is, 20 per cent. Kreutzer[20] made systematic blood pressure measurements in an unselected series of 8,000 children and found 4 cases of coarctation, namely, one in 2,000.

A right aortic arch with a left-sided descending aorta and the left subclavian artery as the last branch has been discussed by several authors as a conceivable anatomic variant.[3, 8, 13, 15] Edwards[8] reported, in 1953, that nine writers had described such cases. Eek,[9] in 1949, stated that a right aortic arch and left descending aorta is a rare variant of right aortic arch; in his series it occurred in only one of 21 cases with a right aortic arch. A right subclavian artery with an anomalous origin, together with a left-sided aortic arch and descending aorta, occurs in 0.5 per cent of all individuals.[16] A mirror image of this condition has been described by Kommerell[17] and by Renander,[19] among others, and in 2 cases by Eek.[9]

The combination of coarctation of the aorta with an anomalous origin of the subclavian artery has been described by Abbott[1] in 2 of the 200 cases of coarctation she had collected from the literature. Another such case was

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Received for publication March 25, 1957.

reported in 1952 by Brodén, Jönsson, and Karnell[5] among 150 cases of coarctation of the aorta known to them. In these 3 cases, the constriction lay between the left and the right subclavian arteries. Stenosis, proximal to the left subclavian, was described by Abbott in 4 of the aforementioned 200 cases. Edwards,[8] in 1953, gave an account of some similar cases from the literature. He emphasized that they must be distinguished diagnostically from cases with constriction of the first part of the left subclavian and hypoplasia of this artery.

CASE REPORT

The patient was a girl, born on Sept. 5, 1949. Both parents and her older sister were healthy. The mother's pregnancy and parturition were uneventful. Birth weight was 3,450 Gm. Operation for cleft palate was performed in 1952. Immediately after birth she had a few attacks of cyanosis. During her first two years of life she had repeated upper respiratory tract infections but no cyanosis. She was physically underdeveloped; at over two years she weighed only 9.3 Kg. and her height was 79.5 cm. Development was otherwise normal.

She had been hospitalized several times, at which times pulsations were found to be lacking in the radial and femoral arteries on both sides, and a faint systolic murmur was present. A tentative diagnosis of coarctation of the aorta was made.

In June, 1955, on admission to the hospital, the following circulatory signs were found: a network of swollen veins was present on the right side of the neck. The cardiac impulse was heaving and displaced laterally. Faint systolic murmur of stenotic type (Grade 2) was present over the aorta. A sharp first sound was audible over the apex (early systolic click). A stronger systolic murmur (Grade 3-4) occurred over the proximal part of the right carotid. The right carotid was markedly hyperactive, the left normal or somewhat hyperactive. Blood pressure (flush pressure): right and left arm, 85 mm. Hg; right and left leg, 40 mm. Hg.

The electrocardiogram showed left ventricular hypertrophy. Pressure measurement in carotid artery was 140 to 150 mm. Hg. Roentgenologic examination of heart (Drs. Kjellberg and Rudhe) showed enlargement of the left side, particularly of the left ventricle. The heart volume was $10.3 \times 6.3 \times 6 = 160$ c.c., corresponding to 240 c.c. per square meter of body surface.

Angiocardiography (Fig. 1) showed normal anatomy of the right ventricle and of the pulmonary artery and its branches. Both carotids were dilated and were given off from the upper part of the ascending aorta. In the midline, the ascending and descending parts of the aorta formed a sharp angle in an oblique plane; consequently, the probable occurrence of constriction in this segment could not be established definitely in the projections used. The descending aorta was filled almost immediately, indicating the presence of a communication in the stenosis. The superior part of the descending aorta was moderately dilated, as in poststenotic dilatation. An outpouching from the descending aorta probably represented the origin of the left subclavian artery. The internal mammary arteries were not dilated, and collaterals, which were narrow, were present only in the soft tissues of the neck.

Diagnosis: Atypical coarctation of the aorta. The constriction was located centrally in the aortic arch, behind and to the right of the trachea and esophagus, between the origin of the subclavian and carotid arteries.

Operation: Oct. 24, 1955 (Crafoord). When the descending aorta was exposed, the left subclavian artery was found to be given off at the usual site, but to be wide and tortuous. The ligamentum arteriosum ran from the same trunk in an anterior direction. Peripherally to this ligament, the subclavian became narrower. The ligament was divided, and dissection was continued in the mediastinum behind the trachea and esophagus, where the right subclavian

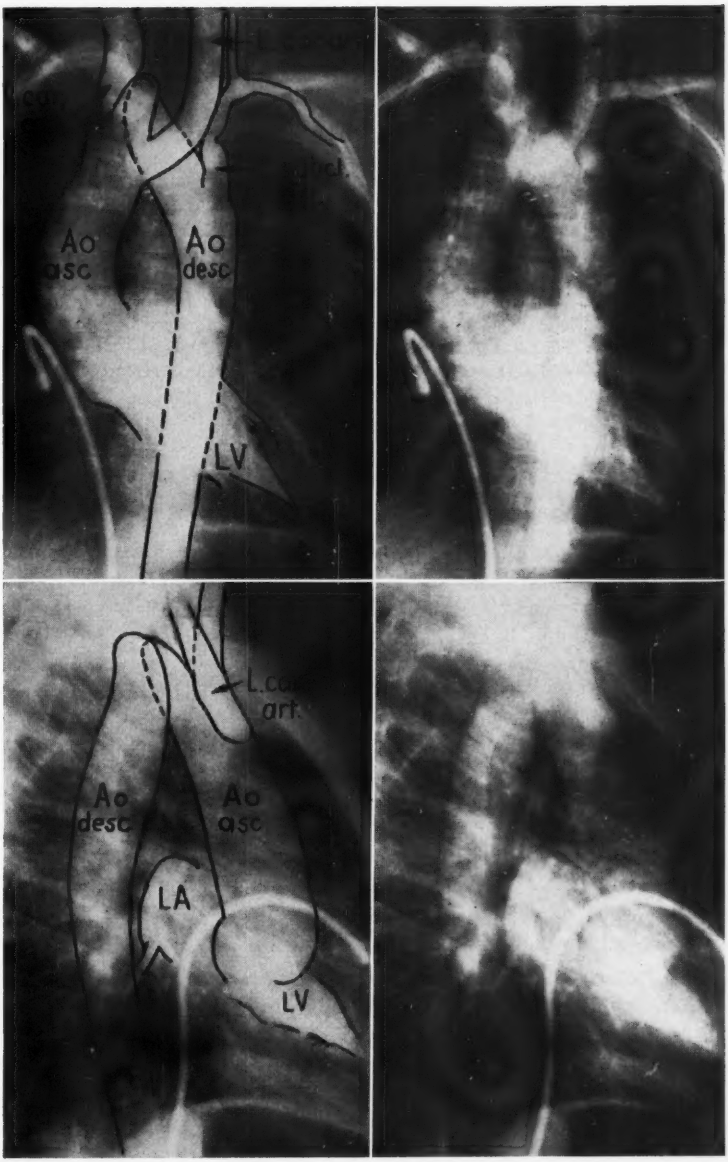

Fig. 1.—Preoperative angiocardiography showing sharp angulation between ascending and descending parts of aorta, dilatation of carotids, both given off from ascending aorta, and diverticulum formation on descending aorta at origin of left subclavian artery.

Ao asc, ascending aorta; *Ao desc*, descending aorta; *L. car. art.* and *R. car. art.*, left and right carotid arteries; *L. subcl. art.*, left subclavian artery; *LA*, left atrium; *LV*, left ventricle.

was found. The distance between the two subclavian arteries was about 5 cm. The constriction was encountered directly proximal to the right subclavian. After splitting the pericardium, the aortic root was dissected on the anterior aspect; the left carotid artery was first encountered, and then the right carotid, almost at the same level. The coarctation reappeared 3 cm. distal to the right carotid. A Potts clamp was applied to the right subclavian, whereupon a palpable pulse appeared in the right arm, and there was increased pressure in the right subclavian artery, peripheral to the clamp. The right subclavian was ligated and sutured. The aorta was then divided centrally to the coarctation, and sutured end-to-end in front of and lateral to the trachea and esophagus.

The patient withstood the operation well. The pulsations in the right radial artery persisted for the first twenty-four hours after operation, but then disappeared. This must be ascribed to the decreased carotid pressure after correction. On discharge, no pulse was palpable in the right radial artery, but the femoral pulses and left radial pulse were distinctly palpable, although the latter was fainter than normally. Pressure: right arm 85/75 mm. Hg, left arm 100/70 mm. Hg, both legs 100/65 mm. Hg.

A follow-up examination was made in March, 1956. The carotid pulses were softer; the pulse in the left arm and both legs was palpable but faint. Pressure measurement in the carotid artery showed 110 mm. Hg, as compared to 140 to 150 mm. Hg, preoperatively. Angiocardiography disclosed a fairly narrow communication between the ascending and descending aorta, and no postoperative aneurysm.

DISCUSSION

The cardinal symptoms in coarctation of the aorta are: (1) absence of femoral pulsations, and hypertension in the upper part of the body; (2) collateral vessels, evidenced by a systolic murmur on the back, in older children and adults palpable in the interscapular region, and visible roentgenologically as notchings on the lower surface of the ribs; (3) in many cases left ventricular hypertrophy on the electrocardiogram; and (4) in some cases, inward bulging of the aorta at the site of coarctation, seen on the roentgenograms.

In the present case, the diagnosis was based on the absence of pulsations in the arms and legs, and on the high carotid pressure. The anatomic details were demonstrated by selective angiocardiography.

The patient's physical underdevelopment, particularly during her first years, can be ascribed to the deficient blood supply. This also applied to her susceptibility to upper respiratory tract infections. After her second year, her condition improved, and it can be presumed that the collaterals then became better developed. There scarcely seems reason to suspect that she had Turner's syndrome (retarded growth, sexual infantilism, webbing of the neck, and an increased carrying angle of the elbow joints). This syndrome has been discussed in connection with coarctation of the aorta, and is said to increase the risk of operation.[7]

The patient was operated on at 6 years of age. Most authors have hitherto considered that operation should be performed at 10 to 20 years,[10, 14] when sufficient collaterals have developed and before arteriosclerotic changes have appeared in the aorta. Boesen and Vendel's aforementioned autopsy series[10] contained numerous cases of coarctation of both the postductal and preductal type. The authors stated that almost 40 per cent of the patients would have been operable; 50 per cent of these infants died before one year of age. In another series of 90 infants reported from Toronto, 60 per cent of the patients

with the postductal type and 89 per cent with the preductal type died before one year of age, most of them even before 6 months.[18] Gammelgaard and Baden[10] therefore concluded that when coarctation of the aorta gives rise to symptoms before 6 months of age, operation should be undertaken immediately.

A tendency to earlier operation can be noted in the literature, the youngest patients operated on being 10 days,[2] 12 days,[18] and 9 months.[10] At our hospital as well, there has been a tendency to operate at an increasingly early age, since it has been found that the aorta and the sutured region grow at the same rate as the individual. For the past seven to eight years, it has been our rule to suggest operation at any time after 3 years of age. In cases with signs of hypertension in infancy, and with electrocardiographic changes, roentgenologic anomalies or clinical symptoms, operation has been advised as soon as the anatomic details have been ascertained. The 2 youngest patients, with coarctation of the aorta operated upon at our hospital, have been 13 and 23 months old, respectively.

Embryology.—The embryologic development of the anomaly in question can be envisaged as follows: In the 2.5 mm. fetus, the dorsal aortae start to fuse upward and downward in the abdominal region, upward to the seventh to fifth intersegmental aortic branches.[6] The ventral aortae become fused to form the truncus arteriosus. Six pairs of branchial arches develop successively between the cranial parts of the ventral and dorsal aortae (Fig. 2, *a*). In the 10 mm. fetus, these branchial arches have already degenerated, with the exception of the third, fourth, and sixth pairs. The third pair becomes the proximal part of the internal carotid artery. The left part of the fourth pair normally forms the aortic arch; its right part becomes partially atrophied and partially included in the proximal part of the right subclavian artery. Its most ventral part becomes the innominate artery. The right and left pulmonary arteries are formed from the sixth pair. Its left dorsal part normally forms the ductus arteriosus (ligamentum arteriosum), whereas its right dorsal part atrophies. The stage shown in Fig. 2, *b* has then been reached. The intersegmental branches from the dorsal (descending) aorta, which form the subclavian arteries, are given off at the level of fusion of the dorsal aortae, but they later shift cranially, so that they generally originate proximal to the ductus arteriosus.

Stenosis and atrophy may occur in other parts of the bronchial arches and aortae. If, with a left aortic arch, this occurs between the right subclavian artery and the right common carotid, it results in a right subclavian with an anomalous origin, given off as the last branch and behind the esophagus. With corresponding atresia, when the right fourth branchial arch instead persists as the aorta, the situation is that shown in Fig. 2, *c*. The left sixth branchial arch then persists as the ductus arteriosus. If, in such a case, the right descending aorta is drawn over to the left and there is, in addition, coarctation between the origin of the right carotid and right subclavian, the conditions are those seen in Fig. 2, *d*, and which correspond to the vascular anomalies in our patient. The collaterals

between the carotid and subclavian regions have also been drawn in this figure. Fig. 3 is a schematic drawing of the topographical anatomy in this case. With respect to the right subclavian, the observations at operation showed that it served as a collateral, and that here the blood flowed in a retrograde direction. As far as the left subclavian is concerned, no data are available regarding the conditions before removal of the coaretation. It seems likely that the blood flowed distally after operation, since pulsations then appeared, and have persisted in the left radial artery.

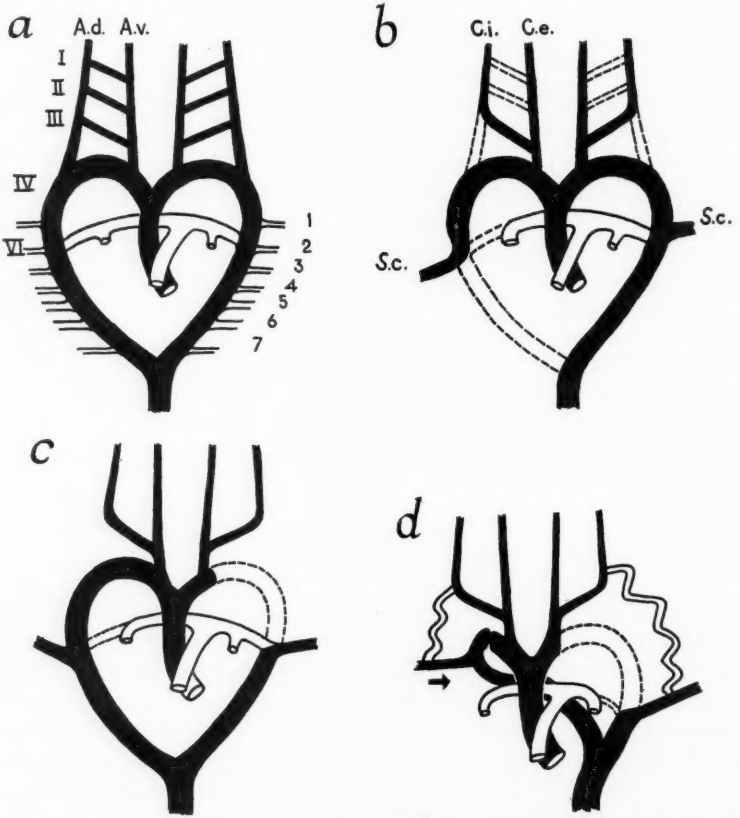

Fig. 2.—*a*, The embryonic stage, when the dorsal aortae fuse to about the seventh intersegmental arteries, and the ventral aortae form the truncus arteriosus.

b, Normal development with left aortic arch and left descending aorta, as well as left ductus arteriosus.

c, Right aortic arch, right descending aorta, left ductus arteriosus, and left subclavian artery with anomalous origin.

d, As in *c*, but with coarctation between right carotid and right subclavian arteries, and dragging of descending aorta to the left. Collaterals between carotid and subclavian regions.

A.d., dorsal aorta, *A.v.* ventral aorta; *C.i.*, internal carotid artery; *C.e.*, external cartoid artery; *S.c.*, subclavian artery; *1, 2, 3, 4, 5, 6, 7*, intersegmental branches of dorsal aorta, of which 5 and 6 become the subclavian artery; *I, II, III, IV, VI*, branchial arches.

According to a theory of Bedford and Parkinson,[4] right aortic arch with left descending aorta is due to one of the three following causes: (1) normal development of a left-sided ductus arteriosus from the left fourth branchial arch; (2) a left subclavian artery developed from the persisting left dorsal aortic root; or (3) a double aortic arch. In all 3 cases, the right descending aorta is stated to be drawn over to the left, behind the esophagus. No support for the theory of the dragging effect of the left subclavian is found in the material described by Eek.[9] In 2 cases, he found a right aortic arch with a right descending aorta, and the left subclavian behind the esophagus, and between the esophagus and the trachea, respectively.

Our patient had both a left-sided ductus arteriosus and a left subclavian artery developed from the persisting left dorsal aortic root. This *may* lend support to Bedford and Parkinson's theory, but does not rule out the existence of some other cause.

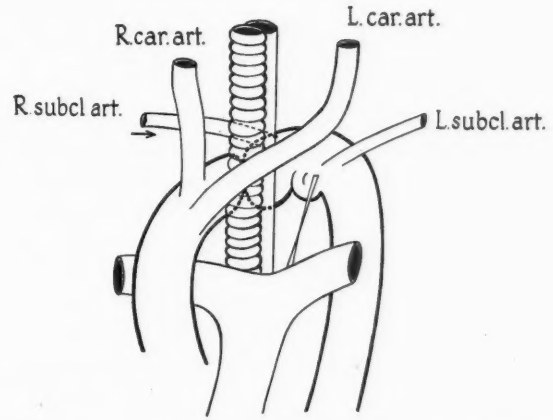

Fig. 3.—Schematic drawing of the topographical anatomy in the case described. The aorta and right subclavian artery run behind the trachea and esophagus. Both carotids are given off from the ascending aorta. The left subclavian starts with a diverticulum formation, from which the ductus arteriosus originates.

R.car.art., right carotid artery; *L.car.art.*, left carotid artery; *R.subcl.art.*, right subclavian artery; *L. subcl.art.*, left subclavian artery.

Both the so-called adult postductal type of coarctation of the aorta and the infantile preductal type are considered to occur at an early embryonic stage,[3, 8, 13, 16] probably during the second month.[3] Our patient also had cleft palate. This malformation is due to a lack of union between the two palatine processes given off medially from the maxillary processes.[6] In the 25 to 30 mm. fetus, the tongue is carried caudally, so that the palatine processes which originally lay beside it come to lie above the tongue. They then grow toward each other and unite in the midline. In the 44 mm. fetus, the formation of the bony palate has already started. Thus, both the vascular anomalies and the malformation of the palate fix the time at which the fetal injury occurred to the later part of the second embryonic month.

SUMMARY

1. A case is described of coarctation of the aorta, proximal to both subclavian arteries, together with a right aortic arch and left descending aorta. The diagnosis was made preoperatively by means of selective angiocardiography, among other examinations. A palpable pulse was lacking in both the radial and the femoral arteries.

2. At operation, the coarctation was resected and the aorta, which ran behind the esophagus, was transposed to run in front of the trachea.

3. The patient, who presented a rare variant of coarctation of the aorta, was operated on at 6 years of age. It is pointed out that the recent tendency is to undertake operation of this malformation at an early age. At the authors' hospital, it is done at any time after 3 years of age. When signs of enlargement of the left side of the heart are present, it is performed even earlier, as soon as the anatomy has been determined.

4. The embryology and pathogenesis of the anomalies in question are discussed.

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BUBBLE DIFFUSION OXYGENATOR FOR CARDIOPULMONARY BY-PASS

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RECENT demonstration of the clinical feasibility of extracorporeal circulation for cardiopulmonary by-pass has opened a new era in surgical treatment of cardiovascular disease.[4-6] Development of an ideal oxygenator for the system has been the major variable in the pump-oxygenator systems which have been described since the pumps which are used are relatively uniform in principle. Several methods of oxygenation of blood have been advocated and most have proved to be satisfactory although some have certain practical limitations. Perhaps the simplest method is the bubble diffusion system in which oxygen is diffused through unoxygenated blood. This method provides for adequate mixing of the blood and oxygen with a large surface area of blood being in contact with the bubbles of oxygen. DeWall and Lillehei[3] have described a simple disposable bubble diffusion system made from plastic tubing and consisting of a diffusion cylinder, defoaming chamber, and coiled helix or reservoir. In our experience, which numbers more than 135 cases of cardiopulmonary by-pass, this device was used in 110 and proved to be satisfactory even for flow rates up to 3 L. of blood per minute.[1, 2] Although the DeWall-Lillehei oxygenator is simple and convenient compared to others in use, certain disadvantages exist such as the expense and availability of the plastic tubing, problems of sterilization, and the necessity for assembly before each operation. Thus, having been convinced of the effectiveness of the principle of the DeWall-Lillehei oxygenator, we devised a stainless steel oxygenator based upon the same principle but which was compact, easily sterilized in the pressure autoclave, and readily assembled at operation.

DESCRIPTION OF OXYGENATOR

The oxygenator consists of four principal parts all made from stainless steel (Figs. 1 and 2). The diffusion plate for oxygen dispersal is perforated by 150 drill holes measuring 0.021 inches in diameter and by a large central opening through which the venous blood enters the oxygenating column (*A*). The column is 75 centimeters long and 3.75 cm. in internal diameter (*B*). At the top of the column, blood bubbles out of the overflow into the defoaming chamber

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This work was aided by a grant from the C. J. Thibodeaux Foundation.
Received for publication April 1, 1957.

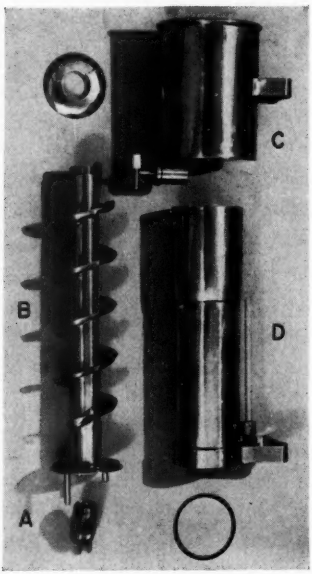

Fig. 1.—Photograph of dismantled oxygenator showing the component parts including the diffusion plate (*A*), oxygenator column with open inclined trough (*B*), defoaming chamber (*C*), and reservoir with level indicator (*D*).

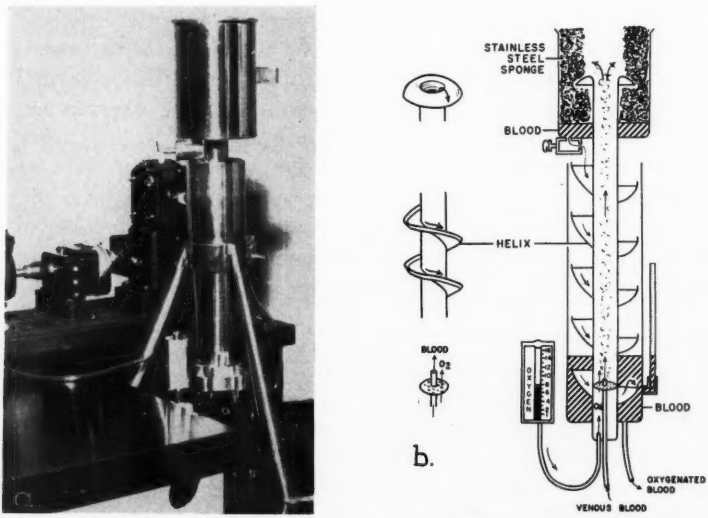

Fig. 2.—*A*, Photograph of assembled oxygenator mounted in tripod holder and connected ready for use. *B*, Cutaway drawing showing method of oxygenation, defoaming, and storage of blood.

(*C*). This chamber measures 15.0 cm. in diameter and contains disposable stainless steel scouring sponges which are coated with Dow-Corning Antifoam-A spray. Here the blood is converted back to a liquid state and leaves the defoaming chamber through a spigot passing onto an inclined circular trough leading into the collecting chamber (*D*). This reservoir has an attached calibrated stand pipe or level indicator made of glass tubing, the inside of which is siliconized. From the reservoir the oxygenated blood is returned to the patient by way of the outflow pump.

A simple method of determining rate of flow is incorporated into the oxygenator utilizing the level indicator gauge on the side of the reservoir. By closing the valve or spigot under the defoaming chamber for a period of 6 to 30 seconds the volume of blood leaving the reservoir may be measured and the rate of flow per minute calculated. Therefore, it is possible at any time during cardiopulmonary by-pass to calculate the rate of flow.

OPERATION OF OXYGENATOR

The assembled oxygenator is removed from the sterile wrapping and placed on a table so that the apparatus is supported on the handles. The inflow tubes of Tygon tubing for venous blood and oxygen are attached to the diffusion plate assembly under the oxygenator and a tube is attached to the outflow from the reservoir. The oxygenator is lifted into the tripod holder on the pump table and secured firmly (Fig. 2). The antifoam spray is applied to the stainless steel shavings in the defoaming chamber. Warm saline at temperature approximately 112° F. is poured into the chamber and fills the reservoir to warm the entire oxygenator and maintain temperature of the blood during by-pass. Shortly before cardiopulmonary by-pass is commenced, flow rate of the outflow pump is adjusted or calibrated using the warm saline in the reservoir and the level indicator. With the reservoir empty, the inflow pump may be adjusted to the desired flow rate by calculating the rate of filling of the reservoir with blood during the priming process. During cardiopulmonary by-pass, the outflow rate is adjusted to the inflow so that a constant level is maintained in the oxygenator reservoir. Fresh heparinized warm blood which has been maintained at body temperature is used to prime the system and fill the reservoir and oxygenating column. Rate of oxygen flow is set at ten times the calculated flow rate of blood, usually between the extremes of 4 L. and 15 L. per minute.

RESULTS

The oxygenator has been used in 25 patients with congenital cardiac lesions, mostly isolated ventricular and atrial septal defects. Detailed results of these operations will be reported elsewhere, but in no instance has the oxygenator failed to provide for more than 98 per cent oxygen saturation of the blood.[2] Defoaming has also been effective as indicated by samples of blood taken along with the tubing from the outflow connection which have been preserved for more than a week after operation and in which no bubbles have been demonstrated. The largest rate of flow used in these young patients was 1,250 c.c. per minute

using a rate of 50 c.c. of blood per kilogram of body weight. With slight modifications of the inclined helix, however, rates of flow up to 3 L. or more per minute should be possible. In no instance have postoperative hemorrhagic complications or excessive hemolysis of blood been evident for periods of cardiopulmonary by-pass up to 30 minutes.

SUMMARY

A stainless steel bubble diffusion oxygenator is described which is highly efficient and has the advantages of compactness, convenience, ease of sterilization, and simplicity of assembly at the time of operation. The device incorporates a simple method of determining flow rate through the system.

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FURTHER OBSERVATIONS ON THE LYMPHATIC PICK-UP OF RADIOACTIVE SILVER-COATED GOLD COLLOID ADMINISTERED INTRATHORACICALLY TO DOGS

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IN A previous publication,[2] this laboratory reported that radioactive silver-coated gold colloid injected into the empty hemithorax of pneumonectomized dogs caused the destruction of the superior mediastinal nodes in 90 per cent of those animals studied. No harmful effects from this treatment were noted. In this communication, these results will be reviewed along with the results obtained by the mediastinal injection of the colloid at the time of pneumonectomy and following the intrapleural administration of the drug at the time of lobectomy.

METHOD

In these experiments, mongrel dogs (weighing 10 to 19.5 Kg.) were used. They were operated upon under aseptic conditions, using intravenous barbiturate anesthesia. At operation or at various times during the postoperative period, radioactive silver-coated gold colloid was injected into the pleural space or mediastinum. The drug was obtained from the Brookhaven pile and processed after the method of Hahn[1] by the Abbott Laboratories of Chicago, Ill. Previous studies[2] showed that it was necessary to use a dose of 40 mc. or more in order to produce a consistent radiologic effect on the mediastinal nodes. The time interval between operation and injection of the isotope did not alter the results. The dogs were sacrificed at intervals ranging from two to forty-four days after injection of the colloid material. At the time of sacrifice, representative lymph nodes were dissected out, cleansed of adventitial tissue, weighed, placed in standard formaldehyde tubes, and the radioactivity determined by a Texas well counter. Histologic studies of the right bronchial, the carinal, the left bronchial, the anterior superior mediastinal, the right superior mediastinal, and the left superior mediastinal lymph nodes were carried out. Previous studies[2] revealed that a significant quantity of the colloid material was present in the mediastinal nodes of dogs sacrificed on the second postinjection day; dogs sacrificed later did not have significantly more gold in the nodes studied.

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Received for publication April 1, 1957.

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Therefore in calculating the node dosage in beta equivalent roentgens, it was assumed that energy emitted after forty-eight hours could be considered as the lymph node dose.

RESULTS

Of the 10 dogs in Group I that received more than 40 mc. of the drug intrapleurally following pneumonectomy (Table I), it is notable that the superior mediastinal group of nodes received adequate irradiation in 70 per cent of the cases. In 2 dogs, the interval between injection and counting was so long that the counts were not reliable. Histologic study of these nodes, however, showed them to be markedly disorganized. If these nodes are accepted as having received adequate irradiation, the percentage is then 90 per cent. There was one death in this group of animals. Dog No. 18 died on the forty-fourth postoperative day of pneumonia which was thought not to be related to gold therapy. This dog's bone marrow was normal. All animals had well-healed bronchial stumps at the time of autopsy.

TABLE I. GROUP I. DOGS RECEIVING INTRAPLEURAL GOLD COLLOID FOLLOWING PNEUMONECTOMY

NUMBER	DOSAGE (MC.)	β* SUPERIOR ANTERIOR MEDIASTINAL	β SUPERIOR MEDIASTINAL LEFT	β SUPERIOR MEDIASTINAL RIGHT
26	44.4	28,546	5,855	13,270
27	44.4	199,254	181,032	315,675
14	50.0	6,211	96	85
12	50.9	88,632	3,451	2,096
8	52.0	198,077	9,869	48,735
9	52.0	83,220	52,440	96,777
10	52.0	34,940	16,783	Not counted
18	75.0	150,314	Count too near background count	Count too near background count
19	75.0	365,489	Count too near background count	Count too near background count
28	150.0	94,620	86,549	181,716

Taken from Matuska et al.: J. THORACIC SURG. 30: 525, 1956, page 527.
*Radiologic effect is calculated as beta roentgen units.

In the second experiment, 12 dogs were subjected to pneumonectomy; with the chest still open, 45 to 51 mc. (in 10 c.c. of solution) of radioactive silver-coated gold colloid were injected into the mediastinum under direct vision. All

TABLE II. GROUP II. DOGS RECEIVING MEDIASTINAL INFILTRATION OF GOLD COLLOID

NUMBER	DOSAGE (MC.)	β SUPERIOR ANTERIOR MEDIASTINAL	β SUPERIOR MEDIASTINAL LEFT	β SUPERIOR MEDIASTINAL RIGHT
5	50	51,700	42,043	48,154
6	51	31,500	16,700	169,800
7	51	15,200	32,000	76,200
8	51	69,000	10,500	25,100
9	49	13,700	2,000	12,900
10	49	76,800	90,400	81,900
11	49	58,000	146,900	76,100
13	50	50,700	299,500	102,800
15	45	109,700	145,800	24,300
18	50	16,500	79,200	54,300
19	50	55,800	8,200	204,500
22	50	40,100	184,000	114,400

of these animals showed histologic destruction of the superior mediastinal group of nodes. Radioactivity was also high, as determined by the Texas well counter (Table II). However, 3 dogs (25 per cent) showed evidence of a bronchial stump fistula.

In the third experiment, 10 dogs were subjected to lobectomy. At the time of operation 35 to 50 mc. of the isotope were placed in the pleural space. The residual lung was inflated and the chest was closed without drainage. These dogs were sacrificed from seven to ten days following the procedure. Nine of the 10 dogs (90 per cent) had significant irradiation of the superior mediastinal group of nodes as determined by the Texas well counter (Table III). On histologic study, all the superior mediastinal nodes showed varying degrees of radiation effect. There was one death in this series. Dog No. 21 died on the fourteenth postoperative day following a left upper lobectomy. At autopsy, pneumonia was present in the left lower lobe and there was atelectasis on the contralateral side. It was felt that this dog died as a result of the procedure, but not as the result of the gold therapy. Except in the dog that died, the remaining lobe on the operated side was free of abnormalities at the time of autopsy.

TABLE III. GROUP III. DOGS RECEIVING INTRAPLEURAL GOLD COLLOID AT TIME OF LOBECTOMY

NUMBER	DOSAGE (MC.)	β SUPERIOR ANTERIOR MEDIASTINAL	β SUPERIOR MEDIASTINAL LEFT	β SUPERIOR MEDIASTINAL RIGHT
12	50	55,400	25,500	19,900
14	50	121,500	251,900	Count too near background count
16	45	81,000	93,100	40,800
17	50	Count too near background count	Count too near background count	6,100
20	50	112,600	110,900	180,100
21	50	194,400	6,300	36,300
23	50	223,800	84,000	256,600
24	50	94,500	26,500	305,200
25	35	177,000	557,000	389,700
26	35	290,000	219,000	384,300

The histologic changes in the mediastinal lymph nodes of all three groups can be considered together. There was total necrosis of the nodes and the immediate surrounding tissues in those instances in which the radiologic effect was the greatest. In the less severe cases, the nodal architecture was partially preserved, but with a complete loss of lymphocytes; plasma cells and reticulum cells were prominent survivors in these nodes. Erythrophagocytosis was noted in lymph nodes with high counts in animals that were sacrificed early. It is notable that the vascular damage frequently assumed a form which strikingly resembled fibrinoid necrosis.

DISCUSSION

One can conclude from these experiments that the lymphatics of the parietal and mediastinal pleurae in dogs drain into the mediastinal lymph nodes bilaterally. These nodes may be destroyed by the intrapleural or intramediastinal

injection of radioactive silver-coated gold colloid. It is felt that if these observations prove to be valid in the human being, a prophylactic type of mediastinal dissection may be added to pneumonectomy in the treatment of patients with resectable bronchogenic carcinoma. Although we do not think it likely that lymph nodes replaced by tumor would pick up sufficient radioactive material to destroy them, it is not unreasonable to believe that small tumor emboli in these nodes could be destroyed by this method. We do not advocate the mediastinal injection of this drug in human beings because of the high incidence of bronchial stump dehiscence in dogs.

Six patients have been treated with radioactive silver-coated gold following pneumonectomy for brochogenic carcinoma at the Thayer Veterans Administration Hospital. No ill effects have been observed from this therapy. Dosage has ranged from 75 to 150 mc. Three of these 6 patients are alive, eight, twenty, and twenty-seven months following therapy, without evidence of recurrent tumor.

SUMMARY

1. Radioactive silver-coated gold colloid consistently produces a radiologic effect upon the mediastinal lymph nodes of dogs following intrapleural injection and following injection into the mediastinum.

2. No harmful effects have been noted following intrapleural administration. The incidence of bronchial stump dehiscence following the intramediastinal injection of the colloid in pneumonectomized dogs was 25 per cent.

3. Currently we are treating human beings with the gold colloid following resective surgery for bronchogenic carcinoma.

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